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(Continuing the Journal of the Allied Dental Societies)

A journal of stomatology; devoted to the advancement and dissemination of knowledge pertaining to the teeth and mouth, and to their relations to the body as a whole

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CONTENTS OF THIS NUMBER

A Summary of the Titles of the Papers, and the Names, Degrees and Professional Relationships of the Authors, With Abstracts or Interpretations of the Original Matter In Each Communication

These abstracts are neither editorials nor criticisms; they do not contain comment beyond the scope of the papers to which they refer. They are intended solely to give the reader a general though clear idea of what the corresponding authors have chiefly to say.

[These abstracts will be reprinted in the December issue, with the title page and other introductory matter intended for bound copies of the volume (IV) for 1922. In the binding of the volume, this insert should be rejected.]

Papers in the Main Body of this Number

A discussion of problems concerning pulpless teeth and pulp devitalization. Authentic information and representative opinions, compiled from responses to a questionnaire. (Third part.)
By Elmer S. Best, D.D.S., Minneapolis, Minn..... 225

Best presents, in this issue, the third of four parts of a compilation of responses to a questionnaire on the general subject specified above. He asked fifty-two questions, all or some of which were answered by the forty-four correspondents named on page 227 (and on the back of the cover) of this issue. The answers to eighteen of the fifty-two questions are presented in this section. Copies of the questions included in the third of the four parts are assembled on page 225, where the general scope of the discussion may be seen at a glance. Best's comment on the replies to each question, as the fourth and concluding part of this study, will be published in the December issue.

(On or about February 1, 1923, the questions and answers, and Best's comment, with any revisions or additions that may be desirable, will be published in book form. The proceeds of the sale of the book will be contributed by Dr. Best to the Endowment Fund of the Journal of Dental Research.)—(W. J. G.)

CONTENTS

A microscopic study of cancellous bone at the root ends of dead teeth. (THE RESULTS OF WORK DONE IN COLLABORATION WITH FRANK E. BLAISDELL, M.D., ASSOCIATE PROFESSOR OF SURGERY, DIRECTOR OF THE LABORATORY OF SURGICAL PATHOLOGY, LELAND STANFORD, JR., UNIVERSITY MEDICAL COLLEGE, SAN FRANCISCO, CALIFORNIA). By JOSEF NOVITZKY, D.D.S., San Francisco, California..... 299

Novitzky states that "our investigations showed clearly the presence of fibrous marrow in cancellous bone at the root ends of dead teeth in all cases examined. This gives us further (in fact, final) evidence of infection in or from devitalized teeth. . . . . It was noted, in all cases examined, that fibrous marrow was most abundant in the *apical* regions of dead teeth and in many instances restricted to such regions, indicating that the septic irritation was apical rather than gingival. The infection, in other words, gains access to the cancellous bone from the dead-tooth dentin by way of the apical foramen and not, as some have thought, from the mouth through the gum flap into the tooth socket. . . . . The presence of fibrous marrow indicated not fleeting but *permanent* irritation, due undoubtedly to reinfection from the dead tooth. This fact is often overlooked—the menace of dead teeth lies not so much in infection as in constant *reinfection*"

The histologic and diagnostic data on which these conclusions were based are presented in detail, and illustrated with reproductions of eight roentgenograms and eleven photomicrographs. These data, and the discussion of their significance, should be studied in detail by all who desire to comprehend the author's position.

"The term 'dead tooth,' as used in this paper," says Novitzky, "signifies every tooth that is really lifeless, namely, *every tooth that is pulpless* . . . . The statement that pulpless teeth are not dead, until the tissues around the root are also dead, might be carried to the logical conclusion that pulpless teeth are not dead until the entire body containing them is dead."

Novitzky sums up the essence of his findings in the deduction that "the condition of the cancellous bone about the root ends of dead teeth offers final and convincing evidence of the fact that dead teeth are dangerous sources of infection, and that in many cases the danger cannot be removed by the old method of extraction."

Novitzky insists that "the deposition of cementum on a dead tooth does not imply that vitality exists in the tooth any more than the fibrous encapsulation of a silk suture in the bodily tissues implies that vitality exists in the silk. The author replanted an extracted tooth *after boiling it*—it was then certainly *dead;* but two years later, when it was removed by operation, sections of it under a microscope showed young lacunated cementum deposited on the old *boiled* cementum" (see the illustrations on pages 316 and 317).

In an addendum, Novitzky discusses a related paper by Merritt (this JOURNAL, 1921, iii, p. 229). Novitzky says in part: "To one familiar with the so-called 'cures' of dead teeth, as seen at operation, any of the eight cases presented by Dr. Merritt would suggest the presence of pathological conditions. A glance at the roentgenograms makes it safe to say that, in each of the cases purporting to indicate apparent restoration to health, a microscopic examination would indicate the presence of fibrous marrow, round-cell infiltration, and dead bone."

Novitzky alludes to the diagnostic unreliability of roentgenograms and suggests that, "in order to demonstrate that an infected pulpless tooth has been restored to health, it is necessary to show (a) that cultures of the cancellous bone around the root end are sterile when incubated; and (b) that microscopic examination of the cancellous bone indicates normal bone and marrow, and an absence of edema and of round-cell infiltrations."

Incidentally, Novitzky adds: "Publication of the evidence in support of the claim of Box, that a tooth has more than one source of nourishment, has been eagerly awaited." (See the next two abstracts for a criticism by Thoma and a reply by Novitzky.)—(W. J. G.)

Comment on Novitzky's "microscopic study of cancellous bone at the root ends of 'dead teeth.'" By KURT H. THOMA, D.M.D., Assistant Professor of Oral Histology and Pathology, Harvard University Dental School, Boston, Massachusetts..... 321

Thoma states that although Novitzky says, "in all cases examined, cancellous bone at the root ends of dead teeth was found to be infected," nevertheless "only one such case was cited and illustrated, the rest of his (Novitzky's) specimens being cases of eburnated bone from supposedly former dental infections. . . . . To substantiate the expectations aroused by the

title of this paper, Dr. Novitzky should present specimens taken from the bone around the apices of pulpless teeth which gave no, or very slight, roentgen evidence of disease."

Thoma regards Novitzky's paper as "an excellent study of regions of eburnated bone in the jaw," and also agrees with Novitzky's "interpretation of the microscopic findings." Thoma also agrees with Novitzky "that new cementum deposited around a pulpless tooth does not indicate that the tooth itself is vital; that the slight amount of nutrition which may reach the root of a tooth through the cementum, as demonstrated by Dr. Box, is inadequate to maintain sufficient vitality in a tooth; and that, after extraction of a tooth, it is necessary to remove the area of diseased bone at the end of the alveolus."

Thoma remarks that Novitzky "has not defined the term 'fibrous marrow'—a term that is not commonly used in pathology," and suggests that "a statement regarding the intended import of this term would . . . . be acceptable to the readers of his paper." (See the succeeding abstract of Novitzky's reply.)—(W. J. G.)

Reply to Thoma's comment on Novitzky's "microscopic study of cancellous bone at the root ends of dead teeth." By JOSEF NOVITZKY..... 323

Novitzky insists that "every pulpless tooth will become infected, if it remains in the jaw, no matter how it is treated. This I took as a proved fact. Further, I took it as a fact that previous investigations had shown infection in cancellous bone at the root ends of all dead teeth. The presence of fibrous marrow was described as final *corroborative* evidence. All the cases presented had histories of infected pulpless teeth. The material consisted of tissue removed by operation in my private practice during a period of some years. The examination included the bone about the root ends of many pulpless teeth in cases that were negative to roentgen-ray examination. Special attention was given to cases that had been 'treated'—cases in which roentgen-ray examination showed what Dr. Thoma calls 'improvement due to treatment, or practically normal conditions around the apex.' Without a single exception, in such cases, the conditions around the apex were not normal; the cancellous bone showed infection. The roentgen-ray examination showed not *improvement* but *change in density;* and change in density

does not indicate absence of infection. A few days ago I removed a large mass of fibrous granulations, most positively infected, although careful roentgen-ray examination had shown no difference between this mass and the normal bone."

Novitzky states that the term fibrous marrow "is used by such men as F. B. Mallory of the Harvard Medical School, and John Fraser of Edinburgh." He presents a statement from his collaborator, Dr. Blaisdell, on the subject of the significance and use of the term.

In conclusion, Novitzky says that "the questions involved are questions of fact—questions that can be answered positively and scientifically without the play of opinion. . . . . For years I have challenged the world to *show one dead tooth*, treated or not treated, that remained uninfected in a jaw six months after pulp devitalization. Now, I suggest that a valuable kind of criticism on the foregoing paper (by himself and Blaisdell) would be that which would point to the *discovery of the absence of fibrous marrow in the bone around the apex of a dead tooth.*"—(W. J. G.)

Further consideration of some of the problems of treatment of pulpless teeth, including remarks on recent criticisms by Ottolengui. By M. L. RHEIN, D.D.S., M.D., non-resident Lecturer on Dental Pathology, Dental School of the University of Pennsylvania; New York City..... 327

Rhein presents a general outline of his present views on root-canal procedures, and of the course of the development of his ideas and methods. He replies, also, to some recent criticisms by Ottolengui. The scope of the paper may be seen, at a glance, in the summary of contents on page 327.

Rhein discusses the preeminent utility of sodium-potassium for the removal of "every vestige of pulp tissue contained within the root canals." He explains the necessity for passing the pulp-removing material through the foramen. The removal of all diseased periapical tissue is regarded as essential, and the usefulness of "electrolytic medication" for this purpose is emphasized. The importance of encapsulation of all foraminal openings, and the particular utility of base-plate gutta-percha and chloro-percha in this relation, are discussed in detail.

Many illustrations, showing successes and failures, are subjected to critical discussion in the light not only of the author's

own views but also of Ottolengui's criticism of some of them in another relation.

The author sums up as follows: "When pulpless teeth are retained in the jaw, they should be placed in such a condition that they can offer no sustenance to microörganisms that may reach them. To accomplish this purpose (a) all pulp tissue must be removed from the canal; (b) all pathogenic conditions of the periapical region must be eliminated; and (c) the entrance of every foramen must be hermetically encapsulated so that there will be no possibility of ingress to the interior of the root. The encapsulation becomes an insulating defensive armament against possible infection."—(W. J. G.)

The basis and administration of advanced standing in schools of dentistry. By FREDERICK C. WAITE, Ph.D., Professor of Histology and Embryology, Western Reserve University, Cleveland, Ohio..... 355

This excellent and timely summary of the basis and administration of advanced standing in professional schools, as applied to schools of dentistry, written by one of the best informed and most experienced observers in this field of education, is so concise, direct, and comprehensive, that, in itself, it is a "brief" on the whole subject and cannot be abstracted effectively without almost complete quotation. The scope of the paper is shown clearly by the "contents" on page 355. A summary of "salient points" is presented on page 366.—(W. J. G.)

The incidence of dental caries in the pre-school age. By RUSSEL F. RYPINS, M.D., Department of Metabolism, Deaner Dental Institute, Kansas City, Missouri..... 369

Rypins believes his paper is the first published record of a study of dental caries "relating to children *under* school age." He noted the incidence of "dental caries that was sufficiently advanced in each case to be obvious to the untrained eye by direct inspection alone. The school districts covered by the report are situated in all parts of Kansas City and represent an excellent mixture of all the grades of human enterprise in child rearing, and of all classes of social distinction."

White children were examined—a total of 1,197. "More than a quarter (27.2 per cent) of the number of these children presented dental caries, averaging over one tooth decayed (1.07) for each mouth examined, and almost four decayed teeth (3.95)

for each of the carious mouths. Of all the teeth erupted, 6.3 per cent presented obvious caries. . . . . In the average child needing treatment, more than four of his teeth would require important surgical attention. In a city of 325,000 population (Kansas City, Missouri, for example), more than 8,000 children would enter the first-grade each year. Over 5,000 of these children would need dental attention, and over 21,000 teeth would require surgical interference. This is indeed an economic problem as well as one of humanity."

The author presents his data, year by year, in a very clear tabular arrangement.—(W. J. G.)

The influence of alkaline and acid tooth pastes upon the activity of salivary enzyme. By FELIX P. CHILLINGWORTH, M.D., Professor of Physiology, Schools of Medicine and Dentistry, Tufts College, Boston, Massachusetts..... 375

Chillingworth reports results with two unnamed tooth pastes. The acidity of the acid paste was "due to acid potassium tartrate." He concluded that the acid paste "inhibited the action of salivary amylase," but that the alkaline paste did not. The author does not suggest any practical applications of his findings. (W. J. G.)

CONTENTS OF THE SUPPLEMENTARY SECTION OF THIS NUMBER

SCIENTIFIC PROCEEDINGS OF DENTAL AND STOMATOLOGICAL SOCIETIES

Harriet Newell Lowell Society for Dental Research, of the Harvard University Dental School

Common tumors that occur about the mouth. By NATHAN CHANDLER FOOT, M.D., Instructor in Clinical Pathology, Harvard Medical School, Boston, Massachusetts..... li

Foot presents a very concise and unusually interesting summary of present knowledge of the subject. Types of common oral tumors are identified and discussed. The author emphasizes the importance to the patient of early recognition of oral tumors by the dentist; and of coöperation between the dentist and physician in this relation in behalf of the patient.—(W. J. G.)

American Society of Oral Surgeons and Exondontists

Annual address of the President of the American Society of Oral Surgeons and Exodontists. By FRANK L. WARREN, D.D.S., Los Angeles, California..... lvii

Warren discusses the past, present (1921), and future. He shows the steps in the development of the Society. He alludes to the "wonderful process of evolution" through which "mouth surgery" has passed "during the last few years," and says: "The fact is that, today, the demands upon the members of this specialty are far in advance of the teachings in the dental schools of this country. Therefore, as a result, what is needed is advanced post-graduate schools with a standard form of teaching the art of modern mouth surgery."—(W. J. G.)

American Dental Association: Committee on Nomenclature

Proposed standardization of some dental terms and phrases. Reported by the Committee on Nomenclature: L. PIERCE ANTHONY, D.D.S., Chairman, Associate Editor of Dental Cosmos, Philadelphia; C. N. JOHNSON, D.D.S., Professor of Operative Dentistry, Chicago College of Dentistry; OTTO U. KING, D.D.S., Secretary of the American Dental Association, Chicago; H. E. FRIESELL, D.D.S., Dean of the School of Dentistry, University of Pittsburgh; and HERBERT L. WHEELER, D.D.S., Professor of Prosthetic Dentistry, College of Dental and Oral Surgery of New York..... lxi

This statement alludes to some of the events that preceded the appointment of the Committee on Nomenclature, and to reasons for the presentation and discussion of the forty-nine words and phrases that constitute the essential feature of the report.

(The recommendations of the Committee are regarded, by this JOURNAL, as good advice, and will be followed by us where they do not conflict with the accepted terminology of the sciences on which dentistry is based. Individual authors will not be *required* by us to abide by the *recommendations* of the Committee, however, for the reason that this JOURNAL was founded to promote freedom of *responsible* and *respectable* expression of truth and opinion in dental affairs without qualification by or for any views its editors individually may entertain. In matters of *opinion*, contributors to this JOURNAL must be free, accordingly, to voice their own views against ours, if and when they so desire.)—(W. J. G.)

BOOK REVIEW

The origin and evolution of the human dentition. By William K. Gregory, Ph.D., Associate Professor of Vertebrate Palæontology, Columbia University; Curator of the Department of Comparative Anatomy, American Museum of Natural History. Pp. 548; 353 figs. Published for the JOURNAL OF DENTAL RESEARCH, New York, by Williams and Wilkins Co., Baltimore.

In the preface Professor Gregory says: "The purpose of the book is to give a brief and concise review of the origin and evolution of the human dentition from the palæontological view point."

The work, we are told, is the result of studies at Columbia University, and the American Museum of Natural History, where since 1900 the author "has had the inestimable privilege of close association with Professor Henry Fairfield Osborn," to whom the book is dedicated.

The writer was peculiarly equipped for the research undertaken by reason of the wealth of material in the museums of America, especially in the American Museum of Natural History, where large collections have been made by successive expeditions in Western America since 1891, resulting in "the discovery of many thousands of fossil mammals from horizons Paleocene, Eocene, and later formations of the West."

In 1883 Professor E. D. Cope, of Philadelphia, discovered "from his studies of trituberculy," that "the tubercular type was ancestral to many, if not all the higher types of molar teeth." This proved to be an epoch making conclusion, which was supported by many other American palæontologists. Dr. J. Wortman, author of "the comparative teeth of the Vertebrata" (1886), writes in 1921 of Cope's theory of trituberculy: "The broad generalization, sought to be established by this theory, is one of the most important and far-reaching deductions within the whole range of mammalian morphology, and is a performance in every way worthy of the great master mind that conceived it."

Cope's theory led directly to the endeavour to trace back of the various bewildering complex patterns of present and past molar teeth to the three-cusped stage. Professor Henry Fairfield Osborn contributed a work in 1907 on "the evolution of the mammalian molar teeth," which supported Professor Cope's conclusions and Professor Gregory claims that the enormous collections of fossil forms recently discovered in America and Europe "confirm Cope's and Osborn's conclusions, that trigonal upper molars and tuberculosectorial lower molars are truly ancestral in pattern." "In 1895 Professor Osborn applied to the cusps of the human molars the system of nomenclature which he had invented at an earlier period for the molars of Eocene mammals, with the simple, and easily remembered terms of protocone, paracone, metacone, hypocone, for the cusps of the upper molars, and protoconid, metaconid, hypoconid, entoconid, and hypoconulid, for those of the lower molars."

Professor Gregory's scholarly work surveys the whole field of verte-

brate evolution, from the appearance of Silurian fishes to the dawn of humanity. Part I deals with the stages of ascent from the Silurian fishes to the primitive mammals of the Age of Reptiles, a fascinating story describing the appearance of the ostracoderms of the Upper Silurian and Devonian, the archaic Devonian sharks, ganoids and lung fishes, and the labyrinthodonts of the lower Carboniferous period. The dawn and development of the great reptiles is next dealt with, showing the origin of the mammal-like reptiles of the Mesozoic age.

The author sketches the appearance of primitive mammals in the Trias, the small triconodonts of the Upper and Lower Jurassic, and the trituberculates of the Purbeck beds, where the continuous changes in the molar forms are evident.

The evolution of the multituberculates is dealt with exhaustively, pointing to their development at a very early date, probably from a separate reptilian origin.

A great blank in our knowledge occurs at the end of the Mesozoic Period, probably meaning "many millions of years," until the Upper Cretaceous, when marsupials of primitive-opossum type appear, probably the ancestors of the modern Australian marsupials.

The author conducts us step by step to the coming of the placental mammals in Paleocene times, and traces their subsequent development and evolution through the Tertiary Age, the orders and families of mammals now existing being the results; and he has applied the tritubercular theory to solve the riddle of the multitude of perplexing molar forms.

The interest increases with the ascent of the Paleocene mammals to the higher primates and the changes, which have taken place in the jaws and teeth of the primates, are clear from lowly lemuroid and tarsioid animals, to the platyrrhine and catarrhine monkeys and to the anthropoid apes, and ultimately to man.

Towards the close of the Cretaceous Period, the great reptiles, which had peopled the earth for vast ages, gradually became extinct, and their places were taken by lowly and primitive placental mammals in the Paleocene Age.

From a study of the large collection of fossil forms of that epoch in the American museums, it is thought probable that a group of archaic Cretaceous insectivores, of the nature of the modern tree shrews (Tupaia) of Eastern Asia, gave origin to the primates.

In so many of the Paleocene fossils the molars were of the Tupaia or tritubercular pattern, and the tree shrew represents probably but little modified the primitive ancestor of the primates. Anaptomorphus, a small form from the Middle Eocene of Wyoming, is then dealt with, a form closely resembling the modern Tarsius of Eastern Asia, showing at that very early period a type which has come down to the present but little changed in its dentition.

Evidence tends to show that the platyrrhine monkeys evolved probably from primitive Notharctus lemuroid forms of the early Eocene. "The stem Platyrrhinæ have many characters of the No-

tharctidæ together with those of certain Eocene Tarsioids," indicating that the two great divisions of catarrhines and platyrrhines separated at a very early period, and evolved on distinct lines, the former attaining their greatest development in the anthropoid apes and man, the latter in the Cebidæ of South America, which are off the lines of human evolution.

The Catarrhinæ probably originated also from an Eocene tarsioid form, but much more highly developed, and of later date than the ancestral form of the New World monkeys.

In the mandible of Parapithecus, a small fossil discovered by Dr. Schlosser in the Lower Oligocene of Egypt in 1913, there was revealed a highly important link in the chain of human evolution. It is the smallest of the Catarrhinæ, with the dentition of i.2, c.1, p.2, m.3, as in the Old World monkeys and man.

Professor Gregory says of it: "Parapithecus is by far the most primitive of all Old World monkeys and apes, and with regard to its well known characters, it may well be regarded as standing in, or quite near to, the line of ascent leading to the anthropoid apes, and eventually to man."

The teeth were symmetrical, the canines did not project, and there were no diastemata, characteristics essentially human, but the condyles of the mandible were widely divergent, the jaw narrowing rapidly in front; the first molar has four cusps, the second and third molars are quinquecuspid; the crowns suggest for the animal a fruit and insect diet.

Professor Gregory then traces the evolution of the Cercopithecidæ, describing the various fossil forms in the Miocene and Pliocene deposits of Europe and Asia.

A very attractive part is the study of the ascent of the anthropoid apes, probably from forms such as Propliopithecus, from the Egyptian Lower Oligocene, described by Dr. Schlosser in 1913.

Of this small animal Professor Gregory writes: "There can be no question that Propliopithecus is an early and relatively primitive representative of the anthropoids."

Hitherto only a small mandible has been discovered associated with Parapithecus in the Oligocene beds of the Egyptian Fayum, the great war putting an end for the time being to further excavation.

One significant characteristic of the dentition is the small size and slight elevation of the canines, so entirely different from those of modern anthropoids. The author considers that "Propliopithecus affords an early stage of the dentition that is structurally intermediate between the lower more tarsioid Parapithecus, and some Dryopithecus-like stage which would be the common starting-point for the main branch leading to the anthropoids and man."

In the Miocene, the next geological period, we meet with the first evidence of the anthropoid apes. Dryopithecus, a large ape closely resembling the chimpanzee, is represented by three mandibles from the Middle Miocene of St. Gaudens in Southern France, and a portion of a similar jaw was found south of the Pyrenees in Miocene beds by Mr. Vidal in 1913.

Hylobates antiquus, an ancient gibbon-like form, was discovered in the same horizon at Sausan in France, demonstrating the distribution of large anthropoid apes at this early period. At the close of the Miocene, the European anthropoid apes entirely disappear, but in the Lower Pliocene beds of the Indian Punjab a large anthropoid ape has been discovered, named by Lydekker "Palæopithecus sivalensis." It is a very interesting form with characteristics uniting it with Dryopithecus, orang, chimpanzee and man. Sir Richard Owen considers it intermediate between the orang and gorilla. Darwin, with his clear-sighted vision on the study of man, "inferred that some ancient member of the anthropomorphous sub-group gave birth to man." Professor Gregory, with the benefit of the accumulated knowledge and vast material collected during the half century which has elapsed since those words were written, remarks that "the detailed studies of the dentition in Part IV of this review leads me to the conclusion that that 'ancient member of the anthropomorphous sub-group' was closely allied to or even identical with either Sivapithecus or Dryopithecus of the modern Miocene Simiinæ, and the comparative anatomical evidence alone is, I believe, sufficient to establish that, in spite of these wide differences in habitus, man is closely allied to the chimpanzee-gorilla group."

Lastly, Professor Gregory deals with Pithecanthropus, a human precursor of the Pliocene or Lower Pleistocene of Java, and conducts us to the study of the low human type of the Heidelburg man. It is known by the massive mandible found by Dr. Shætensach in the gravels of the River Mauer, near Heidelburg, representing a very low and primitive type, but essentially human. The Piltdown remains, Professor Gregory considers, are not human, but more probably those of a female chimpanzee.

With Mousterian and Aurignacian man it is not necessary to deal in this review. Professor Gregory's book will be found a storehouse of information on the subject, being the outcome of long and patient study, and will be welcomed as a valuable addition to our knowledge.

The facts are clearly arranged and described; the fine series of photographs, drawings and diagrams perfectly illustrate the narrative; the story becomes absorbing as gradually unfolded. The book numbers 548 pages with 353 illustrations.—Review reprinted from the *British Dental Journal*, 1922, xliii, pp. 1025–1028.

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The responses to Dr. Elmer S. Best's questionnaire (JOURNAL OF DENTAL RESEARCH, 1920, ii, p. 285; June), which was intended "to obtain a consensus of current opinion and practice relative to problems concerning *pulpless teeth and pulp devitalization*," are published, in part, in this issue (see page 225) and will be presented, in an additional part, in the December number. The parts will be assembled, revised, and issued as a book. Forty-four eminent dentists have collaborated with Dr. Best in this work. The price of the book will probably be about $4.00.

The author of each of these two valuable books has very generously agreed to present to the Endowment Fund of this JOURNAL the net proceeds of the sale of his book and the JOURNAL will publish each volume as the financial beneficiary accordingly.

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In order to facilitate the editorial and scientific work of this JOURNAL, a complete dental library will be needed. Contributions to such a library, which this JOURNAL is now assembling, of literary and historical matter in dentistry and stomatology, of any and all kinds and of any era (from clippings to books), are solicited from friends of this JOURNAL. Duplicates that may be received can be profitably used in suitable exchanges with other libraries. Shipments may be addressed to this JOURNAL at 437 West 59th Street, New York City, where the library now occupies a small room reserved for it in the Biochemical Laboratory. Acknowledgments of gifts to this library will be made in this JOURNAL. Publishers of dental journals and books are requested to favor this JOURNAL with copies of their publications, for the furtherance of dental research through the development of this permanent library.

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See a review of the book, on pages 9 to 12, preceding.

WILLIAM J. GIES

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A Palæontological Review

BY

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Curator of the Department of Comparative Anatomy, American Museum of Natural History, New York; Associate Professor of Vertebrate Palæontology, Columbia University

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Authentic Information and Representative Opinions, Compiled from Responses to a Questionnaire[1]

(*Third Part*)[2]

ELMER S. BEST

Minneapolis, Minnesota

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[1] Best: *Journal of Dental Research*, 1919, i, p. 523; 1920, ii, p. 285; 1922, iv, pp. 1 and 129.

[2] The *first* part was published in the *Journal of Dental Research*, 1922, iv, p. 1 (March); the *second* part, p. 129 (June). The four parts of which this study consists will be issued in book form, on or about February 1, 1923.

[3] See the "introduction," on page 2, of the *first part* (March issue).

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I. NAMES, ADDRESSES, AND IDENTIFICATION NUMERALS OF THE CORRESPONDENTS, WHOSE ANSWERS ARE GIVEN IN SECTION II[4]

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7.[6]
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11. Davis, W. Clyde, University of Nebraska, School of Dentistry, Lincoln, Neb.

[4] See the prefatory note, on page 5, of the *first part* (March issue).

[5] A question mark, at the head of a response, indicates a reply from one of the unidentified correspondents. See foot-note 6.

[6] After the manuscript had been sent to the printer, it was discovered that there were no identified responses from four of the correspondents named on this list (6, 7, 34 and 44). It was then too late to rearrange the identification numerals throughout the text, without prohibitive expense. The names of the four correspondents have accordingly been eliminated from the list, but the corresponding numerals (which are not used in the text) have been allowed to stand in the list. See foot-note 5.

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45. West, John B., 306 West Church Street, Elmira, N. Y.

II. COPIES OF THE QUESTIONS IN THE QUESTIONNAIRE; WITH THE ANSWERS, TO EACH, ASSEMBLED IN THE ORDER OF THE IDENTIFICATION NUMERALS ON THE LIST OF CORRESPONDENTS IN SECTION I[7]

Questions XXXV–LII

XXXV. *In case the dentine of a non-vital tooth is not infected, and is surrounded by or encased in living cementum, which in turn is surrounded and nourished by a normal vital periodontal membrane, would you consider such a tooth a foreign body? Will it become infected, and will nature try to throw it off?*

(1) No; it is still nourished by the periodontal membrane surrounding it. It will remain healthy, and nature will not throw if off.

(2) No; it is simply a pulpless tooth. It may become infected, but clinical evidence does not show that pulpless teeth in otherwise healthy condition are exfoliated earlier than teeth having pulps.

(3) No. It will in all probability become infected eventually, but nature will not attempt, at least for many years, to throw it off, unless the tooth is affected by pyorrhea alveolaris.

(4) No—to each item.

(5) Such a tooth, the occurrence of which is practically impossible, is partly dead tissue, or partly a "foreign body." While I believe such a tooth may become infected, nature probably will not throw it off, because the tooth is nearly surrounded entirely by vital periodontal membrane.

(8) Not a "foreign body." Not necessarily infected later or thrown off.

(9) Absolutely no.

(10) I would not call such a tooth a foreign body, because it is able to fulfil every physiological function, and produces no pathological condition. A tooth with a vital pulp, the crown of which is partially replaced by a filling, may be said to be a living organ supporting a foreign body. The non-vital non-infected dentine, in a tooth such as the one described, may be considered simply as a filling substituted for the normal dentine of the root, just as gold may be substituted for

[7] See the prefatory note, on page 5, of the *first part* (March issue).

the dentine of the crown. Such a tooth may become infected, and so may a tooth with a vital pulp. Nature will not attempt to throw it off so long as it remains in the condition described in the question.

(11) No, most emphatically. You are wrong when you ask this question about a non-vital tooth. This is a *vital* tooth; no doubt you refer to pulpless teeth.

(12) No; but I'd remove the non-vital tissue and fill the canal, whether infected or not.

(13) No. It should not become infected, if the canals are properly filled; and nature will not attempt to throw it off, unless the periodontal membrane becomes seriously involved.

(14) I do not consider it a foreign body; do not believe it is likely to become infected; and do not believe nature will try to throw it off.

(15) I would not consider such a tooth a foreign body. Nature will not try to throw it off so long as the root is aseptic. The pulps of sound teeth occasionally become infected and die. In such a case, we must suppose that infection of the pulp is secondary to a focus elsewhere in the body. The apical tissues of a pulpless tooth may similarly become infected.

(16) I do not consider such a tooth a foreign body any more than I do a tooth with lifeless enamel. I do not believe it will become infected, neither do I believe nature will try to throw it off.

(17) A non-vital tooth is only half-dead, the pericemental tissues remaining as a source of nourishment to the dentine. Altho there is a region of diminished resistance at the apex, a tooth such as that described in this question should not be looked upon as a foreign body. It will probably resist infection for a long period, and nature will not try to throw it off.

(18) I would not consider that the tooth as a whole, up to the time it became infected, was a foreign body. If, as the question indicates, the cementum is still living and receiving nourishment, I would not treat that portion of the tooth as a foreign body. If, however, as your question implies, it becomes infected, it should then be treated as a foreign body, for, if no treatment is given to it, nature will make the same effort to relieve herself of the tooth as she does of any other foreign tissue, except that because of the mechanical and physiological nature of the body under consideration, the process will be very slow. (See my answer to question XLI.)

(19) No. It can become infected, but I do not think nature will try to throw it off.

(20) I believe not.

(21) No. It will not be infected so long as all external means of infection are precluded.

(22) No; I do not consider such a tooth a foreign body. If properly treated, it is no more apt to become infected than any other tissue; and, if it is used, nature will not try to throw it off.

(23) Nature will tolerate it always, provided infection does not find its way to the apex. But it probably will, unless mechanically and chemically prevented. If you mean: will infection go through dentine to the cementum, I would say, I do not think so; but I do not positively *know*. I have not seen anything that appears to indicate exfoliation, or the non-toleration of cementum, even when dentine *is* infected.

(24) No.

(25) Microscopic examination of apical tissues sometimes results negatively with the best available technique, so it would seem that in such cases the tooth is not a foreign body. I cannot say it will become infected, but it may.

(26) No, and it will not necessarily become infected. We have all seen many cases where this has not happened.

(28) No—to each item.

(29) Not being able to determine whether or not the dentine in a non-vital tooth is infected, I would have no such vexatious problem to annoy me. (No statistics.)

(31) I do not. Not necessarily. I do not believe nature will exfoliate such a tooth if proper antagonists are in situ.

(32) About "fifty-fifty." If it is treated properly, then filled with oxychloride of zinc (except at the immediate apex), nature will "tolerate" it; but nature will be suspicious of it always, and in time—say from ten to thirty years—will "stand it up against the wall to be shot." In the meantime, it will have helped to grind a good many grists.

(33) Emphatically, no. A tooth is not a foreign body just because the pulp has been removed. Nor does nature treat it as a foreign body. Did you ever observe nature trying to get rid of a sequestrum: she *never lets up for an instant* until she has succeeded in what she set out to do, namely, to eliminate the dead bone. She doesn't act this

way at all in the case of the pulpless tooth. She does act that way in the case of an acutely abscessed tooth *for a time.* Dentine is composed principally of inorganic matter, and inorganic matter does not die, in the sense that we usually think of death, which sense is that of putrefaction and disintegration. Even supposing, however, that the dentine is partially dead, with the cementum still alive, the junction between life and semi-death is *within the tooth itself;* so why should nature act on a surface of living cementum as though it were "a piece of dead bone" or "a foreign body?" She doesn't.

Speaking of cementum, it is a pretty-well established fact that hypercementosis occurs more commonly on pulpless teeth than on vital teeth. Isn't this evidence that nature attempts to seal infection hermetically within the tooth? Isn't nature here trying to keep intra-dental infection *intra-dental?*

I have lately obtained three radiographs that show the protective efforts of nature most beautifully. First, is a radiograph showing the recession of the pulp and the heaping up of dentine between the pulp and an encroaching carious cavity. Second, is a case of hypercementosis in which nature is trying to seal infection in the tooth, to prevent its escape into the bone. Third, is a case showing a circle of osteosclerosis surrounding a granuloma, nature here again trying to seal up the encroaching infection, within a limited region, to prevent its metastatic distribution in the body. In the case of the granuloma, nature builds both the protective fibrous wall of the granuloma, and the surrounding wall of denser bone.

(35) Such a tooth is a desirable member of the dental arch, and no more subject to infection than any other therein, provided its roots have been successfully filled.

(36) To my mind, such a tooth is favorable for retention. I have seen many teeth of the incisor group, which became non-vital through trauma, remain apparently uninfected for a long period of years.

(37) *If* the canal work is properly done, the danger of infection is remote.

(38) No. So long as these conditions prevail, the tooth is not a foreign body; and, if the canal operation is efficient, the tooth will not become infected. There should be no greater tendency to exfoliation than for other teeth.

(39) (a) Certainly not, if the root has been *aseptically* filled, which the question presupposes. Who can say? (b) Healthy tissue has an ample defence against infection. (c) Nature always combats infection. The presence of a chronic condition indicates a balance between opposing forces, resistance and infection. Only a *dead* patient fails to react to injury; there is always an effort to "throw off."

(40) According to the latest research work (Davis), such a condition seems to be highly desirable, and is probably the safest in which a devitalized tooth may exist.

(41) *No.*

(42) I would not. All pulpless teeth have a tendency to degenerate, as any one who has practised for a number of years has observed among his own patients. The crowns become opaque and more or less darkened in time; but I have observed that, as long as there is a normal vital periodontal membrane and the tooth is kept in function, nature does not try to throw off the tooth. If such a tooth was disinfected and the roots filled before infection penetrated beyond the root, the tooth is not likely to become infected afterwards. I have observed some such cases over a period of between thirty-five and forty years, that continued to function well, the individuals continuing in perfect health.

(43) I do not consider this type of tooth a foreign body, providing the canal or canals can be aseptically and accurately filled. Authorities seem to conclude that most pulpless teeth are "positive" when a bacterial-culture test is made of them, no matter how satisfactory the root-canal filling may seem to be radiographically. The question then arises: were these teeth filled aseptically?

(45) If the cementum is built over the apical end of the root, so as to seal the foramina, I should not consider the tooth a foreign body. If that condition does not exist, and the apical end of the root is not encapsulated, it is quite possible that the tooth will become infected.

(?)[8] No. It may become infected; if so, removal is usually necessary.

XXXVI. *Do you believe in treating diseased apical tissue through the canal, or do you believe in surgical removal of such tissue?*

[8] A question mark, at the head of a response, indicates a reply from one of the unidentified correspondents. See foot-notes 5 and 6.

(1) This has been asked in several of the previous questions I have answered. In the case of granuloma on any of the anterior teeth, when it is very desirable to save them, I advise surgical removal of the apical end, as I have stated in detail, in preference to any treatment through the canal. I believe some cases of apical infection can be cured by pumping phenol into the canal and sealing it up for a few days, but it is the only remedy I know that will go beyond the apex and destroy infection.

(2) Through the canal first. If failure results or if there are complications, treat surgically.

(3) Through the canals ordinarily. For severe cases, both.

(4) I believe in trying to treat first through the canal, when time permits.

(5) There are three methods of treating the diseased apical tissue: first, by routine root-canal treatment, with or without ionization; second, by this first method and surgical removal of such tissue; third, by surgical removal of the teeth and diseased apical tissue. The last mentioned gives the highest percentage of successes.

(8) Through the canal in incipient cases only. All others should be treated surgically.

(9) Through the tooth, if possible; if not, then by the surgical route.

(10) In cases where treatment of periapical disease is indicated, it is generally wise not to depend alone upon treatment through the canal, but to establish surgical drainage. Surgical removal of such tissue is not essential unless root resection is indicated.

(11) In acute cases, treatment through the canal suffices. Chronic cases demand surgery.

(12) Ionism or surgery. I am skeptical of resection.

(13) Treat moderately through canals with palliative medicaments, attempting to give nature a chance to take care of these tissues. Resort to surgical removal where palliative measures have failed, and retention of teeth seems desirable.

(14) I believe in surgical removal of such tissue.

(15) If it is not too extensive, through the canal. If the infection resists such treatment and a part of the cementum is necrotic, then resection of the root is indicated, the resistance of the patient being considered.

(16) If you refer to infection, I must say that I do not believe in either method of treatment.

(17) Diseased apical tissue cannot be permanently eradicated by treatments through the canal. Symptoms may be alleviated and comfortable function restored, but we have no means of determining that all infection has been destroyed or that the trouble will not again return after a period of time. Extraction, followed by careful curettage, is the best and surest method of removing apical disease.

(18) I believe treatment through the canal is the proper method; but, if the area is extensive and the tissue is destroyed beyond the possibility of repair, or if there is any small particle that is liable to remain and act in the nature of a sequestrum, then I believe surgical treatment is the proper method.

(19) At present, I believe that treating diseased apical tissue through the canal offers the greatest percentage of successes.

(20) Treat through the canal, and remove surgically only as a last resort.

(21) Both; if the canal is large enough to enable one to get to or through the foramen. The canal must be sterilized as far toward the apical tissues as possible before surgical removal of these tissues is attempted.

(22) It depends on the nature of the case. I should first treat through the canal. If my treatment was not as effective as desired, I should open through the gum. I should curette only after drainage and washings had proved ineffectual.

(23) Treat through the canal when possible. When not possible, the other method is the only hope. Surgically, it looks good; bacteriologically, it seems to look bad (see my answer to question XXI). Practically, the patients don't like it.

(24) In some cases where the apical disease is not extensive, the tissue can be made healthy by treatment through the canal, if the canal can be opened. In other cases, I believe in surgical removal of such tissue.

(25) I do not treat diseased tissue through the canal. Surgical removal of such tissue is the one chance of curing the lesion.

(26) I should not resort to surgical removal until canal treatment had failed.

(28) Surgical removal. Sometimes you may force medicaments through the canal and break up the infected tissue.

(29) I *believe* in treating such a tooth through the canal. At times I might try surgical procedure, without much faith in it! (No statistics.)

(30) I believe in being conservative in such cases, and advise treatment through the canal first. If this does not give satisfactory results, surgical removal may be indicated if the tooth is to be saved.

(31) Both, depending on the case, age of the patient, clinical history, etc. No definite rule can be prescribed.

(32) "Depends." If diseased tissue can be removed without excising the root end, do so; then file the root-end smooth, and treat and fill the canal.

(33) Surgical procedure, where it can be applied, is to be preferred as a means of treating periapical disease. Ionization, I believe, is preferable to ordinary medication through the canal of the tooth.

(35) Apicoectomy is the safer and more reliable procedure, although fraught with uncertainty as to permanent results. When apical infection is extensive, extraction and socket curetting are indicated.

(36) I believe that in certain cases of diseased periapical tissue, under favorable conditions, such tissue is amenable to treatment by way of the canal, especially where the agent used is dichloramin-T. In a great majority of cases of diseased apical tissue, aseptic surgery is indicated. I do not believe in a long series of treatments to determine the efficacy of the therapeutic procedure in a given case. It is very hard, by radiographic examinations, to "check up" accurately on the progress of such drawn-out treatments. In the very few favorable teeth of this class, the results of my treatment have to be fairly prompt and definite.

(37) I believe that where there is a secondary involvement, surgical removal is by far the wiser procedure.

(38) Through the canal, if the involvement is not extensive and there is no evidence that the root is necrotic. In other cases, if treatment is indicated, it should be surgical.

(39) I believe both methods are at times successful. Go through the canal, when the lesion is one of recent development, is acute and not too extensive, and the treatment causes no irritation. The alternative

is surgical removal of the pathologic tissue. Extraction is usually successful in removing periapical disease.

(40) In cases of *brief duration*, where a diseased condition of the periapical tissue is merely a reaction to the diseased condition of the pulp, treatment through the canal is successful in a large percentage of cases and should be attempted under favorable conditions (state of health of the patient). In cases of *extended duration*, and in each case where the root apex has become absorbed, surgical treatment is indicated. When symptoms of inflammation of the pulp are detected early and the pulp is removed, conditions for treatment are more favorable than when the pulp has been infected for a long time (chronic inflammation of the pulp, or necrosis). Pathological examination of teeth with diseased pulps in situ shows that inflammation of the pulp occurs before bacteria have actually invaded it, probably due to toxins that reach the pulp through the dental canals. If, on the other hand, the pulp has become infected, and has been in this condition for a long time (usually a condition of pulp necrosis or gangrene), the dentinal tubules all along the root canal are invaded by bacteria.

(41) Treat as best you know how the apical tissue. If it does not respond to the treatment, try removal of the apex. We have had successes and failures by both methods.

(42) It depends upon the extent of the disease. Many cases can be successfully treated through the canals; others require surgical removal of the affected apical tissue before there will be resolution.

(43) Surgical removal of such tissue seems to be the more positive.

(45) I think much the safer procedure is surgical removal, although I have treated diseased apical tissues by ionization.

XXXVII. *Do you consider that a diseased tooth with infected dentine is more liable to have a deleterious effect on the surrounding tissues, even though the canal was cleansed of the infected pulp, than would be the case if the symptoms of inflammation of the tooth pulp were detected early and the pulp removed before it had become infected?*

(1) I certainly do.

(2) Yes.

(3) Yes.

(4) It depends upon the size of the tubuli, the number of foramina, and the height from the apex of the foramina. It is always preferable

to avoid infection, and infected dentine is certainly more liable to have a deleterious effect than non-infected.

(8) Yes.

(9) Yes.

(10) With the greatest skill and care, an infected tooth may frequently be rendered safe; but early removal of the pulp, and conservative filling of the canal under aseptic conditions, offer greater security.

(11) Yes; however, the difference is not great, as the osmotic current ceases when the pulp canal contains nothing of a fluid or semi-fluid nature.

(12) Yes.

(13) I would say it is *less impossible*, rather than "more liable." If the canal is properly sealed throughout, I believe the factor of liability becomes infinitesimal.

(14) I do.

(15) Yes.

(16) My opinion is that a tooth with long-standing infection of the dentine cannot be sterilized without injury to periapical tissue. Therefore, I believe the removal of an inflamed pulp will be more successful.

(17) Yes; and such a tooth should be extracted. Long-standing infection of dentine is almost impossible of complete sterilization, and a tooth so infected should be looked upon as a menace to the surrounding structures.

(18) Most certainly; if the pulpal inflammation can be detected before the pulp becomes actually infected, the effect on the surrounding tissue is decidedly better. An effort should be made, every time, to determine whether or not a pulp is infected. The effect on the surrounding tissue is extensive in many cases, to the degree of the infection that causes the disturbance.

(19) Yes.

(20) Yes.

(21) Yes, absolutely.

(22) Probably early removal of a diseased pulp is preferable; nevertheless, I believe that infected dentine can be so treated that it need have no deleterious effect on the surrounding tissues.

(23) It does not seem to me that infection of the dentine is important if it can be disinfected before apical tissue is affected seriously. If you mean by way of the cementum, I would say that I do not think so; but then I do not know that anyone *knows* anything about it. How is one to tell whether the dentine is infected near its periphery, even if laboratory tests prove the canal sterile? If you have a vital pulp to begin with, you assume sterility, or at least not as much infection; but Price claims that apical infection follows mere pulp exposure and there you are again! Personally, I act as if I did not believe it. Now surely isn't it worth a million?

(25) I do so believe.

(26) Possibly, but not probably, except from a theoretical standpoint.

(28) Yes.

(29) I give this one up; too deep for me. (No statistics.)

(30) Yes.

(31) If dentine is infected from the periphery, where cementum is denuded, it will always remain a possible source of injury to surrounding tissues. If dentine is infected from the pulp canal, and the canal is thoroughly disinfected, I believe, from the results of certain experiments by Marshall, in the University of California, College of Dentistry, that infectious organisms become walled in and are destroyed, do not pass outward through the cementum, and hence cannot be a source of injury. I believe any inflamed pulp will prove to be infected, except where trauma has occurred.

(32) I do.

(33) Yes; certainly the prognosis is better when an inflamed pulp is removed than when an inflamed pulp has been allowed to die and become infected before its removal. In the case of an inflamed pulp, if aseptic precautions are observed, as they most emphatically should be in all pulp-canal work, there is no great amount of infection in the canal to overcome. In septic pulps, there is a tremendous amount of infection in the canal and dentine to be overcome.

(35) Of course, due to the fact that infection may be lurking in dentinal tubuli and cemental lacunae, even though the root-canal work is perfect, the tooth is the safer factor if its canal treatment and fillings were done before the pulp could become infected.

(36) Yes.

(37) Not having studied this question, nor having seen any statistics on it, I prefer to leave it unanswered.

(38) Yes, but infected dentine without other complications is not irremediable.

(39) Yes, *more liable.*

(41) Yes.

(42) Yes.

(43) Yes, I do; because, when the dentine once becomes infected, it is most difficult to render it sterile.

(45) Yes. I do not think argument is possible on the proposition that a vital tooth, from which the pulp was removed under surgical aseptic conditions, is safer than one that already has the dentinal tubules infected with microörganisms.

XXXVIII. *To what extent do you consider x-ray evidence final in determining the occurrence, extent, and nature of apical pathological conditions?*

(1) I think an x-ray picture, while not always *positive evidence* in determining the presence or absence of apical infection, is absolutely necessary. Without it we could determine nothing.

(2) It is final only in well marked and definite cases, which must be and usually are supported by clinical findings.

(3) One of the most valuable aids to diagnosis, but should always be verified before operating, as many mistakes have resulted from the fact that the operator placed too much dependence upon x-rays; in fact, made his diagnosis solely by this means.

(4) I consider x-ray evidence final and conclusive only when the radiogram is sufficiently plain to indicate attachment of the darkened region to the apical portion, and when the lamina dura is attenuated or cannot be traced by the aid of a strong magnifying glass. X-ray evidence is accepted as final only when it is an exact, life-like, reproduction of the field covered, and not camouflaged. Dark areas are taken to represent either organic matter or space; and light areas to indicate calcium, metal, or anything that obstructs the rays.

(5) One must not forget that an x-ray examination is only part of a general examination, and is most valuable when supplemented by the findings of such other examinations. While we may assume that the

apical periodontal membrane of a devitalized tooth is infected, although the apical area appears normal on the x-ray picture, still we are not at liberty to make the latter diagnosis from the x-ray picture alone. On the other hand, a perceptible apical area on the x-ray picture may represent a region that may be sterile for a long time after successful root-canal treatment and ionization. Furthermore, in my experience, the vast majority of cases of cysts were sterile. Under no circumstances is one permitted, in this relation, to make a diagnosis of abscess, apical or otherwise, about the jaws, because the existence of pus cannot be detected by means of the x-ray picture; and an abscess means a circumscribed collection of pus. I am not speaking, however, of sinus conditions. In other words, while an x-ray examination is of great value, it must be checked by clinical findings. It is obvious, therefore, that the radiodontist, whose specialty consists of making dental radiograms and "reading" them, oversteps the boundaries of his specialty, if he gives advice regarding treatment. He should be permitted only to *interpret the film* and his diagnosis should be made from the film only, as in the case of the *general* radiographer.

(8) Never final. Radiographic evidence is relative in value only, and is frequently very misleading.

(9) I regard the x-ray as a splendid diagnostic aid but not as infallible.

(10) Radiographs with sufficient definition may be relied upon to disclose the presence of periapical disease. The extent of the pathological condition is often obscured because of the superimposition of radiopaque structures. The nature of an apical pathological state can be determined by a careful study of its radiographic outline. The radiolucence caused by granulomata blends gradually into the normal bone shadow. In primary cases the radiolucent area is circumscribed, and in advanced cases it is diffuse. The radioparency caused by a radicular cyst is circumscribed in outline and is sharply defined from the surrounding normal bone shadows by a radiopaque line. An acute alveolar abscess may present no radiographic evidence or may be a secondary infection from a granuloma or cyst.

(11) There is nothing final about any one means of diagnosis. The x-ray is only one important link in the chain of evidence.

(12) I do not consider it final, only suggestive or strongly indicative.

(13) X–ray evidence should be considered simply as one means of diagnosis. I would hardly call it final in any case, unless it were confirmed by other evidence. It is certainly unwise to base a diagnosis on x-ray evidence alone.

(14) I consider the x-ray a very valuable aid in determining the facts regarding such conditions.

(15) The x-ray alone is not usually sufficient to determine the extent of apical disease. The x-ray affords only a shadow picture and the shadows are frequently deceptive. Often several exposures at different angles are necessary to get a correct representation of the true condition. The physical appearance of the overlying tissues is often helpful in determining the condition. The sharp steel probe is also helpful in some cases.

(16) A good radiogram will even show pathological conditions of the periodontal membrane.

(17) We have no better means than the x-ray of determining the presence of apical disease. Most teeth that are chronically infected at the apex do not present local symptoms. Referred pains, or other symptoms, indicative of toxic absorption, may be elicited, but we have to resort to the x-ray in order to locate the diseased teeth. In order to determine the exact nature of apical disease, bacteriologic study is necessary, but we are not so much concerned about the nature of the infection as we are about its detection and eradication. We can determine its nature during or following the operation of removal, if we wish to go farther with our treatment than eradication of the apical foci.

(18) Except in cases of gross disease, I cannot say that I believe the x-ray is "final" in any pathological condition. At best it is only confirmatory, as a rule. That does not detract, however, from its value as a diagnostic aid; but the point I wish to make is that it *is* an *aid*, and that all reasoning on the extent and nature of the disease surrounding the apices of teeth must have its origin in the knowledge of pathology and histology. There are, of course, many gross things on which the x-ray must be considered "final," such as a large cyst or a large granuloma; but, as a rule, existence of such things can be determined even without the help of the x-ray. Therefore, while it is very likely

the greatest single aid that we have, in determining conditions about the apices of teeth, yet the x-ray must never be relied upon absolutely as "final" in pathological conclusions.

(19) I do not consider it final in all cases, but it is by far the most reliable evidence available.

(20) In a case where radiographs show complete destruction of bone, I regard this evidence as final so far as the periapical bone is concerned. So far as the apex itself is concerned, disease can be definitely determined radiographically only where erosion of this structure has taken place.

(21) Ninety-five per cent of "occurrence;" eighty-five per cent of "extent;" seventy-five per cent of "nature."

(22) The x-ray is valuable in practice but it has its limitations. We can usually detect loss of bone and bone regeneration by it. If properly used, it shows the curves and length of roots. But many "areas" that appear in radiograms have no clinical foundation. Goadby showed slides at a recent national dental meeting (1920) that brought out this point. You cannot detect the presence of pus, or of infection, with the x-ray. Many "areas" do not represent diseased tissue but *cicatricial* tissue. I never rely on the x-ray alone in determining the presence or absence of apical disease.

(23) If a granuloma *shows*, it is there—infection at least, perhaps an abscess. If a cloudy effect shows, infection probably is present. If nothing shows, bad canal filling and putrescence may be present; in a few cases, even septic apical pericementitis. There must be a time when an infection *begins* to produce an abscess or granuloma, and when the *showing* is so slight as to be indistinguishable. Can the patient absorb infection and have systemic infection at this point, or even before? It is agreed that he can absorb from a granuloma and thus acquire systemic infection. Shall we say positively that "*nothing showing*" means a harmless tooth? How then shall we decide—by logic, symptoms or radiographic appearances? The radiograph may give positive proof *but not negative proof*.

(24) Radiographic evidence should always be considered with clinical evidence. Teeth often show x-ray evidence of a slight degree of apical disease when clinically the pulps are vital. In cases of extensive apical bone rarefaction and absorption, it is often possible

to base diagnosis on radiographic findings alone. Frequently one can say, pre-operatively, whether one is dealing with a granuloma, an abscess, or a cyst.

(25) The x-ray is only an aid in diagnosis. A tooth should not be condemned on its evidence alone, but many cases need hardly be classed as doubtful. Frequently, extraction has proved that I am unable to determine the occurrence, extent, and nature of apical disease.

(26) To the extent that it shows a suspicious area, or confirms a suspicion I may have.

(28) X-ray evidence is not absolute—only an aid in diagnosis.

(29) I do not consider the x-ray *final* in any such case. The ray, in *conservative* hands, is only an aid in diagnosis; taken by itself it is valueless as far as pathology is concerned. (No statistics.)

(30) I believe that the x-ray is the most efficient aid we have for determining the occurrence and extent of apical disease. It is an aid in determining the nature of the same, though the microscope is a necessary adjunct.

(31) I never consider it "final," and accept it only as an aid in diagnosis.

(32) Very useful, but not positive. I am very partial to a good electric mouth lamp.

(33) X-ray evidence is by far the best evidence we have of the existence or non-existence, and the extent, of apical disease.

(35) Radiographs show bone destruction but cannot show it until it proceeds beyond a root apex, if it started on a buccal or lingual side. Intensity of infection may be measured by "time and extent" of bone destruction, but small foci may escape radiographic detection for the reason stated above. Bone destruction indicates staphylo-streptococcal infection, but not the exact nature of the bacteria involved. Systemic resistance must be taken into account.

(36) It is a tremendous help, but not dependable in the absence of bone disintegration—not dependable on the extent of the destruction and the nature of the involved organism. It is grossly dependable when it gives a definite outline of the boundary of the bone destruction, lamina dura, etc., when determining what disposition the surrounding tissues are making of the invading toxins. It gives absolutely no evidence on the virulence of the infection.

(37) This much is certain; all that the x-ray reveals as rarefied is certainly pathological or, to say the least, is not normal. But, unfortunately, the x-ray does not reveal some conditions that are *abnormal.*

(38) When correlated with clinical evidence, it is final in proportion to the skill applied in securing the evidence, to the experience and judgement in interpretation, and to the anatomical limitations presented. The average standard of dental radiography is so inferior that teeth are condemned and exonerated upon the evidence of radiographic examinations which are inadequate and often misleading. Radiodontic interpretation is the common field of practice for dentists, physicians, specialists, osteopaths, and chiropractors, and a popular pastime for the laity. With this condition prevailing, the "factor of error" is evident and must be considered. In some cases, anatomical structures greatly restrict the information which can be obtained from a radiodontic examination.

(39) X-ray evidence is valuable in confirming clinical findings. When destruction of tissue is extensive, as revealed by radiolucent areas on the film, the only fact actually established is that there "appears" to have been destruction of bone. It is incorrect to say, solely from the evidence of the film, that such tissue is infected, for the film may show what "appears" yet actually "is not." Infection and disease may be determined histologically. The clinical findings including the x-ray are valuable. Where all the findings concur, it is proper to accept the evidence as established fact based on knowledge.

(40) I believe that roentgen evidence of apical infection is positive and final. In some cases, however, it is necessary to take several pictures from different angles, especially in multi-rooted teeth, to get a true picture of the condition around all the roots. Only very good roentgenograms should be used for diagnosis. I know that many dentists who have taken up roentgenology, lately, very often produce extremely poor pictures, both from photographic and roentgenographic points of view. If these pictures are for one's personal use only, the temptation to be satisfied with a poor result (on account of lack of time, etc.), is very great. I believe that the use of such inferior pictures, as a basis for important diagnoses, is more frequent than anyone realizes.

Negative results in a roentgen examination are not always final. In making a diagnosis of periapical disease and in deciding what is indicated by the different conditions, as they appear on the roentgenogram, the following should be considered. Variations in the appearance of so-called "abscess cavities" in roentgen films have, to my mind, more to do with their anatomical location than with their pathological content. The defect in the bone consists, as is evidenced by examination of skulls, of a cavity caused by bone destruction. The loss of bone is what is registered in a roentgen picture: the size of a dark area around a tooth apex, therefore, depends on the size of the abscess cavity; the difference in shade and texture of its picture depends upon its location. If the outer or inner cortical plate of the bone has become perforated, we get a deeper shadow than from a cavity in the cancellous part of the bone between the two thick, unaffected, cortical plates of the mandible. Again, if the apex of the root is close to the surface, as is often the case with upper incisors, we find only a shallow depression in the surface of the bone. This would give a very indistinct picture, and the structure of the underlying cancellous part of the bone could probably be distinctly seen in the area on the film. In the upper bicuspid and molar region it is important to keep in mind the fact that often there is only a thin layer of bone over the tooth apices, especially if the latter protrude into the maxillary sinus. The bone is not sufficiently thick for an abscess cavity to be formed and, therefore, we usually get very little roentgen evidence of the infection.

(41) I do not consider x-ray evidence final in determining any but the most apparent cases. It is only a help and should be used only in connection with clinical examinations.

(42) Anything but final.

(45) I do not consider it final at all. The roentgenogram shows only two surfaces of the tooth; and a small radiolucent mass may exist on either of the two surfaces without showing. In fact, in operations for the clearing up of focal infections, I have discovered comparatively large labial masses of destroyed bone that did not show in the roentgenogram at all. I think that clinical evidence must be used, in every case, in interpreting the pathological conditions shown by a roentgenogram.

XXXIX. *In orthodontic treatment, what causes pulp death during the process of moving the teeth and how can that cause or causes be eliminated? What do you do or advise when pulp death occurs for this reason?*

(1) I would not send one of my patients to an orthodontist who did not have the skill, equipment, and judgment to move teeth without endangering their vitality. I never killed a pulp, although I have done lots of work along this line. I would consider it malpractice to kill one.

(2) Too rapid movement, or traumatism due to faulty occlusion, generally. If the root is fully developed, or nearly so, and the case is anatomically favorable, I treat and fill. I have radiograms of one such case, a lateral incisor, showing completion of the apical third about four years after the root was filled.

(3) Too rapid movement, or too much stress on individual teeth. If death of the pulp occurs, remove it, unless the canals are not sufficiently formed to make filling feasible. In that event, remove the tooth.

(4) Pulp death from orthodontic treatment is due to interference with the circulation of blood to and from the tooth at the apical foramen. Should it be the pulp of an undeveloped tooth, extraction is imperative.

(8) (a) Probably caused by pressure thrombosis. (b) Remove the pulp, and fill the canals if possible.

(9) Too rapid movement of the tooth. I have never had such a case; but if it occurred, I would try to sterilize and fill the root.

(10) This is a question for orthodontists to answer. I do not know. I do not recall having had a case of this nature to treat, but suspect that, if the condition were discovered before extensive periapical involvement, I should attempt treatment.

(11) Too rapid movement may result in a thrombosis. This does not occur with expert orthodontists. I am not an orthodontist.

(12) Move the teeth very slowly, or death of the pulp may occur. Apply the usual treatment.

(13) Too rapid or too forcible movement superinduces excessive inflammation and strangulation of the pulp. Remove the pulp, if the root is complete; otherwise, remove the tooth. (Use better judgment thereafter.)

(14) Owing to the fact that I refer all such cases to an orthodontist, I have no data on this subject.

(17) I am not practising orthodontia. I am not competent to answer this question.

(18) Pulps die in orthodontic cases, as a rule, as a result of too rapid movement of a particular tooth (when not due to actual decay). I can conceive, however, that a misplaced band, being too far root-wise, would set up an inflammatory process that would result in devitalization of the pulp. Traumatism, as a rule, is the cause of death of pulps; traumatism that results in interference with circulation about the apex of the tooth, producing a stasis or sluggish circulation, and finally an obstruction. Traumatism may account for infection through the periodontal membrane and pulp death may result from autoinfection. When the pulps die as a result of orthodontia, I retain the teeth just the same.

(19) Too rapid movement seems to be the cause. I handle a pulpal involvement, under this condition, as I would whether orthodontic treatment were being given or not.

(20) In the scientific practice of orthodontia, pulp death, during the process of movement, is extremely rare. Unskilled orthodontic treatment may be the cause of pulp death. When such occurs, the root canal should receive most careful treatment in order to preserve the affected tooth. Orthodontic treatment is sometimes blamed for destruction of pulps, when in reality, injury is a result of some minor accident. The teeth of children are frequently injured from falls or blows received during play, and devitalization of the tooth is not discovered for some time afterward.

(21) Too rapid traction of teeth, too much stress upon them, resulting in impairment and sometimes total loss of their blood supply. Move the teeth more slowly, giving nature a chance to rebuild tissue behind the roots. If detected within three months after the pulp is devitalized, remove the pulp, sterilize the canals, and fill.

(22) The cause of pulp death, during the process of moving teeth, is serious disturbance of the tissue about the teeth. This is the result of too rapid movement or too rapid torsion of the teeth. Proceed as in any other case of pulp death.

(23) It is caused by an aseptic arterial hyperemia ending in aseptic venous hyperemia, stasis, death. Open aseptically and treat as though a living pulp were present, i.e., pulp removal, antiseptic sedation, root filling. Bleaching after root filling may be necessary to overcome the suffusion of venous hyperemia.

(25) Anemia and the general physical condition are real factors. A cause sometimes is the orthodontic appliance, used as a machine for the purpose of pushing teeth, instead of as a therapeutic agent for the purpose of applying a stimulant. When pulp death has occurred, I treat the tooth as I would any trauma case.

(26) Too rapid movement, and not sufficient rigidity in the appliance on the teeth that are being moved.

(28) Strangulation. I have handled many cases, but never had a dead pulp from this cause. Rough and unskilled handling, and neglect of the patient, are causes.

(29) The death of a pulp, due to orthodontic treatment, can only occur through incompetence. My advice would be to change the orthodontist. (No statistics.)

(30) Trauma. I would advise removal of the pulp in such cases.

(31) Refer to orthodontists.

(32) I have never noticed death of pulp from moving teeth for their regulation. Of course, such an accident may be prevented by moving them slowly. (I have seen two cases of pulp death from the use of the Perry separator.)

(33) In orthodontia, pulp death occurs most frequently from moving a tooth, losing that movement, then re-moving the tooth, and keeping this up, until an intense inflammation results. Pulp death may also occur from moving teeth too rapidly, particularly in the mouths of the older orthodontic patients. When the pulp dies, treat as in any other case of pulp death, that is to say, ionize, or resect the end of the root, or extract; do whatever is indicated for the particular case.

(35) Strangulation is the cause. Teeth should be moved slowly enough to avoid disturbance of blood supply to the pulps. Treatment: germicidal, apical root-canal filling, with encapsulation preferred to "shyness."

(36) I should judge that this would be caused by too radical a movement of the tooth, but I have never seen such a case. The later

appliances are very mild in their action, and I cannot see how this can occur. Should it occur, I would extract the tooth, if, by retaining it, the health of the patient, or the life of the tooth itself, were endangered.

(37) Too rapid movement. In youths, extract.

(38) Caused by the application of incorrect or excessive traction, which may be avoided by conservative and careful orthodontic treatment. If the death of the pulp occurs before the development of the tooth is complete, the removal of the tooth is indicated. An efficient canal operation should be performed, if conditions are favorable.

(45) In my limited experience, with such cases, I think the pulp is devitalized by too rapid movement of the teeth. This can be obviated by having the appliance under perfect control, and moving the teeth very slowly. If the root-ends were complete, I should attempt a pulp-canal operation.

XL. *Do you think one should differentiate between teeth that contain infected pulps and teeth from which healthy pulps have been removed, in determining whether or not non-vital teeth may be retained?*

(1) Yes, a tooth that has once been infected is quite different from one that has not been infected.

(2) Not necessarily, if present conditions are similar.

(3) Again, it depends upon the health of the patient and one's ability to fill all the canals.

(4) I would give preference to teeth where no infection had occurred, unless satisfactory reconstruction of the mouth depended upon the formerly infected teeth, which I would then render aseptic and healthy.

(5) In a case of serious illness, no differentiation should be made between devitalized teeth, while in any other case dental treatment should be given a fair trial first.

(8) No, providing that the infected canals may be properly cleansed, sterilized, and filled without infection of the periapical tissues. The chances for success, however, are greatly in favor of the second class.

(9) Yes, decidedly. It is very difficult if not impossible to sterilize dentine from which a putrescent pulp has been removed, while, if the pulp has not been infected, an aseptic root-canal filling can be made with relative ease.

(10) The condition of the periapical tissues should be the deciding factor in the extraction or retention of teeth. If it were a question of elimination of one of two teeth, all other conditions being equal, I should certainly extract the tooth which had been infected.

(11) You are not drawing the line of distinction at the proper place. This line should be drawn between cases where the end products of fermentative decomposition have appeared and those where the end products have not. Apply treatment, provided all cementum is vital.

(12) The presumption is in favor of the tooth from which healthy pulp was removed—if the fact of healthiness is known.

(13) I see no reason for differentiation, if only the pulp tissue is involved.

(14) I do.

(16) Yes, whenever possible.

(17) In my judgment, teeth that contain infected pulps should be extracted. If radiograms do not reveal evidence of apical disease about teeth from which healthy pulps have been removed, or about teeth in which the canals are properly filled, the teeth should be retained.

(18) It is always good reasoning to give the preference, in the retention of devitalized teeth, to one from which a healthy pulp has been removed; but, if you can conclude that you have absolutely sterilized the tooth and made a perfect root filling, there is no reason why a tooth with a previously infected pulp should not be retained, providing there has not been too much destruction of the periapical tissue.

(19) Yes.

(20) Yes. I believe there is a much better chance of keeping teeth free from infection, from which healthy pulps have been removed, than there is of sterilizing infected teeth.

(21) Yes, surely.

(22) No.

(23) It is purely a question of whether one can disinfect infected teeth. After that they are equal in the race for acquisition of bacteria. Perhaps I am wrong in thinking that a devitalized tooth, with *empty* tubules, stands the better chance theoretically for absorption of antiseptics like iodoform to keep out infection.

(25) I do. The tooth from which a healthy pulp is removed may be considered more favorable, as the tubuli of the dentine once infected may not be entirely sterilized permanently.

(26) No.

(28) Yes.

(29) Whether or not non-vital teeth should remain or be extracted must be determined from their actual present condition. The previous history of the pulps need not be considered. (No statistics.)

(30) I think one should differentiate between these two types of teeth. The former was an infected tooth originally. The latter may or may not have become infected.

(31) Not necessarily. Either type of operation may be a success or failure.

(32) I do.

(33) I *do* think that one should differentiate between teeth that contain infected pulps and teeth from which healthy pulps have been removed, in determining whether or not pulpless teeth may be retained. Where a healthy pulp has been removed, if aseptic surgery has been practised, the case has never been infected. In a case of septic pulp, much infection existed and may or may not have been overcome.

(35) Take systemic conditions into account first. If disinfected as much as possible (sterilized), then germicidally and apically filled pulpless roots, which contained infected pulps, are safe—with high systemic resistance. Properly treated and filled roots are safe after healthy-pulp removal, even with low systemic resistance.

(36) Yes, the only difference being that the one containing infected pulps is *probably* infected, whereas the tooth from which the healthy pulp has been removed, if not infected, is a *candidate* for infection.

(37) No. As far as "we" know, neither is deleterious to the system.

(38) Although the future of teeth from which healthy pulps have been removed is more favorable, the decision should be based upon other factors.

(39) The determining fact is the establishment of the presence of disease. If one or the other class is diseased, preference should go to neither, so far as extraction or retention is concerned, provided extraction is indicated. Infection does not "come from the tooth itself;" it arises from invading bacteria. These may "come" (a) by

way of the pericementum, by an extension of a lesion; (b) by way of infected dentine, due to an infection of the pulp; or (c) may have a hematogenous origin. It is believed that the latter condition does not arise in the absence of an injury to the periapical pericementum (such as may be caused through a traumatic occlusion), and when the associated structures are not diseased. Tissue to become infected must be injured or abnormal to a degree that permits organisms to cause disease. It is not enough that bacteria reach certain structures; this they doubtless do continually. What *is* important is the fact that there they may proliferate and cause disease.

(40) I think such a history is important in determining whether the teeth should be retained.

(41) Certainly the *dentine* in a tooth from which a healthy pulp has been removed is less liable to be infected than would be the case if the pulp were left until pulp degeneration and infection occurred, and this question would largely enter into the decision to remove or retain such a tooth.

(42) Yes.

(45) I don't think it would make much difference unless one knew that the root-canal operation on the tooth from which the healthy pulp was removed was a surgically aseptic one. This information would be very desirable, provided root-canal work was standardized and every dentist kept accurate case records.

(?) Yes.

XLI. (a) *In correcting a case of malocclusion, do you first make a complete x-ray examination and vitality test of all the teeth in order to locate the unerupted or non-vital teeth?* (b) *If you make an examination, partial or complete, what do you do with the non-vital teeth present?* (c) *Do you complete your realignment with them in place or do you advise their early extraction?* (d) *What do you advise in case the third molars are impacted?* (e) *What do you think of the alveolus surrounding non-vital teeth as compared with that surrounding vital teeth?*

(1) As I stated before, I believe it better practice to relieve the patient of all non-vital teeth or unerupted teeth, and replace them with skilfully constructed fixed bridges or with removable bridges properly made. There is no difference in the alveoli surrounding a non-vital tooth and a vital tooth, provided there has been no infection about the tooth and it has been properly filled.

(2) I do not practise orthodontia.

(3) Use the x-ray to locate missing teeth or suspected pulpless teeth, although a complete x-ray examination would be preferable, and would, I believe, be of greater assistance to the orthodontist, particularly in determining size, length, and position of roots. Leave non-vital teeth, unless they are beyond proper treatment. Extract them immediately, unless they are necessary for anchorage in the correction of other teeth. Extract impacted third molars. The condition of the alveolus surrounding a non-vital tooth depends on the extension of apical involvement from the tooth.

(4) (a) Yes, if there is anything to indicate the probability of unerupted teeth being present, or if there is any doubt as to the vitality of any of the teeth. (b) If all conditions are favorable for the thorough cleansing and filling of the canals and the tooth, there being no indication of systemic involvement, I advise their retention; otherwise, their extraction. (c) If it is determined that a non-vital tooth should be extracted, I prefer to have it done at once, and to complete the correction of the case afterward, moving other teeth forward, if the age of the patient and other conditions permit, thereby closing the space. (d) I advise their extraction. (e) Seems slightly more resistant to the movement of the teeth, unless there has been considerable absorption.

(9) I do no orthodontia.

(10) I am not an orthodontist and therefore refrain from answering this question.

(11) We make complete x-ray pictures of all mouths, upon which we expect to do orthodontic work, in order to determine the position of all erupted as well as non-erupted teeth and to locate, if possible, any regions of infection. In the case of pulpless teeth (we do not use the term "non-vital" in this connection): their movement is attempted as in the case of teeth with vital pulps. However, subdental tissues about such cases are closely scrutinized during the process. In case third molars are impacted and the orthodontia contemplated does not indicate the liberation of this impaction, the impacted teeth are removed as a preliminary procedure. On item (e) let me say: it depends entirely upon present or past infection. If the pulp has been removed from the tooth under an anesthetic, and that removal or

partial removal has not been accomplished entirely to the apex, and if such a case has not gone through the process of inflammation, the periapical space will be lessened and the bone made more dense about the apex of the pulpless tooth than it is about the apex of a vital tooth. Again, if there has been marked infection at some previous time and the case has made a good recovery, the periapical region will be made up of scar bone, which is more dense and less liable to a secondary infection than normal bone. But if the tissues of the pulpless tooth are, at the time of inspection, undergoing inflammatory changes, attempts to move the tooth will only aggravate the condition, and the loss of the tooth is most probable.

(12) I do not practise orthodontia.

(13) I refer such cases to a dependable orthodontist.

(14) See my answer to question XXXIX.

(17) I do not practise orthodontia, and am not competent to answer this question.

(18) An x-ray examination and a vitality test of all the teeth are made before starting a case of orthodontia. No particular difference is made for non-vital teeth—I put them in alignment as I do the rest. Some trouble, however, is anticipated with them. There is always some inflammation attending every case of orthodontia, and that inflammation is increased for non-vital teeth. In the case of impacted third molars: if we have reason to believe they are liable to make trouble either from pressure or infection, or if they interfere with proper regulation of the case, they are removed. If not, they are allowed to remain.

I think this answer affords a good opportunity to comment on the question whether or not a non-vital tooth is a "foreign body." If, in the regulation of non-vital teeth, the inflammation becomes intense and the periodontal membrane infected, nature herself will treat that organ as a foreign body. If, in a case of orthodontia, nature so treats a tooth, then the extraction of that tooth is advised. I do not think there is a particular difference (and I presume any difference would be physiological, surely not anatomical), between the alveolar process surrounding a non-vital tooth and that of a vital tooth, except in a case where there is actual apical infection. (See my answer to question XXXV.)

(19) (a) Yes. (b) The orthodontic treatment has no bearing on the decision, or on the handling of such teeth. (c) If we believe we can treat them satisfactorily, we leave them in place. (d) Whether or not we remove them depends upon whether or not we believe they are doing, or will do, injury to the second molars. (e) In the absence of infection, we believe them the same.

(20) In undertaking the correction of a case of malocclusion, I do not think it necessary in all cases to make a complete x-ray examination and vitality test of the teeth. However, when there is the least suspicion of the existence of non-vital or impacted teeth, an x-ray examination should be made. If non-vital teeth are found, they should receive proper treatment and be retained, unless they are considered a menace to the health of the patient. I believe that many non-vital teeth may be saved by skilled treatment. When third molars are impacted, I advise their extraction. The alveolus surrounding a non-vital tooth is apt not to be as healthy as that surrounding a vital tooth, particularly where the inflammatory process has been severe or prolonged.

(21) (a) Yes. (b, c) Extract and realign. (d) Remove impacted third molars. (e) The alveolus may be physiological or pathological when in contact with a non-vital tooth. It may be partially decalcified and entirely healthy.

(22) (a) I make a complete x-ray examination when there are unerupted teeth. (b, c) If non-vital teeth show no signs of disturbance about them, I proceed as if they were vital. (d) I remove impacted third molars. (e) I presume that the alveolus is much the same in both cases.

(23) I do not do orthodontia. I wish I did nothing else.

(25) We do not correct—we treat. Nature does the correcting. A full x-ray examination is not necessary in all cases. The problem of the non-vital tooth has been discussed in answers to previous questions. Third-molar impactions cannot be considered as a class. Each one is an individual case requiring special consideration.

(26) (a) Yes. (b) Treat and save, if possible. (c) Complete realignment, "with them in place." (d) Extract. (e) No known difference, providing there is no infection present.

(29) (a) As there is no known way of *positively* determining whether the pulps in certain teeth are alive, except by drilling into them, this would not be done for a prospective orthodontic patient. (b) Non-vital teeth would be treated as vital teeth, if their conditions so warranted. (c) Each case would be a study of its own. (d) Remove them. (e) I do not *think* of it. An ample supply of trouble is always on hand without this. (No statistics.)

(31) Referred to orthodontists.

(32) I do not now practise orthodontia.

(33) I do not consider my opinions on these questions worth giving: "I leave it to the orthodontist." However, I can see, of course, the advantage of making a complete and careful examination of the mouth before starting work, and that this examination might include radiographs and application of the electric test for pulp vitality.

(35) I know nothing of orthodontic technique and pathology, because for years I have limited my work to periodontia and stomatology. No examination of a mouth is worth making at all without complete radiographic and vitality tests, the former if only to learn bone and diploetic conditions, and the latter to guard against surprises or serious errors. Systemic resistance and intensity of infection should govern the removal or retention of non-vital roots—expecting more of the former in the child, due to the fact that leukocytes in the arterial blood reach infected spots more actively.

(36) I do not make a complete x-ray examination or vitality test in such a case, except where there is obvious doubt as to the vitality of a tooth. In cases of abnormally large teeth, depending somewhat on the age of the patient, we look for third molars and, if impacted, we remove them, preferably after the seventeenth year. We rarely extract a vital second molar as a remedy for the impaction. In a case of non-vital bicuspids or molars, we advise their extraction, especially of a lower first molar or upper first molar, where there has been any history whatever of infection. If nature has successfully warded off an acute infection, I feel that the alveolus was made denser by nature's efforts.

(38) "A complete radiographic examination and vitality test" is essential to orthodontic treatment. The disposition of pulpless teeth is decided by existing conditions, but they are not condemned solely

because they are pulpless. The removal of impacted third molars is advisable when it is believed they are an etiological factor in malocclusion or other disturbances. The alveolus around a pulpless tooth is rarely abnormal unless infection is present.

(40) Complete x-ray examination seems to be very desirable, and it should always be *complete*.

(45) I do not do orthodontia, but refer such cases to a specialist. I think that third molars, if impacted, should be removed. The alveolus surrounding a non-vital tooth does not seem to be as dense or as resistant to pressure as that around a vital tooth.

XLII. *In your opinion, is dentine infected by preparing cavities for fillings and then allowing some time to elapse before placing the filling, no precaution being taken to seal the cavity in the interim and to prevent admission to it of saliva?*

(1) I certainly do and I never leave a cavity unprotected one hour if I can help it. I always put in a gutta-percha or temporary stopping of some kind, and soak the cavity with clove oil before I do that. It is good practice to line the cavity with a varnish composed of chloroform and rosin, and then to insert a temporary filling, when you prepare a cavity for an inlay and have to wait half a day, or a day or two, before you can make and insert it. In the preparation of abutments for bridges, it is always necessary to protect the cavities this way; and for jacket crowns, put on a temporary crown and seal with chloropercha.

(2) It may be.

(3) Undoubtedly.

(4) It has not been my experience that it does to any appreciable extent. It depends on the length of the period of exposure. I have never filled a cavity that had been exposed without mopping it out with phenol, which I believe is sufficient for all practical purposes after the cavity has come in contact with saliva.

(8) Yes, and the pulp as well, in many cases. However, if the dentine is sound, such penetration by infectious organisms is not deep, as a rule, and these organisms can be hermetically sealed in the dentinal tubuli with the adhesive filling materials.

(9) Yes.

(10) I feel convinced, from clinical evidence, that such a procedure often results in infection and death of the pulp. In my opinion, most pulps which are said to die of thermal shock really die from this cause, or from failure to remove or sterilize all infected dentine. A treatment of the dentine immediately after cavity preparation, with a silver-reduction solution, followed by a temporary filling of eugenol and oxide of zinc with cotton fibres, will prevent subsequent thermal shock or other evidence of pulpal complications.

(11) I believe all exposed dentine should be protected at the earliest possible moment, provided the tooth contains a vital pulp, as the osmotic current is in the direction of the pulp.

(12) Yes—always cover cavities temporarily.

(13) Yes, but hardly to any great depth, unless an unreasonable period of time has been permitted to elapse.

(14) Yes.

(16) My opinion is that many pulps are infected in this manner.

(17) Dentine is probably always infected secondarily from an infected pulp. However, leaving open and unprotected a prepared cavity is very poor practice—almost malpractice.

(18) The dentine is most assuredly infected, and the mistake is commonly made. No doubt many inlays would give better service if the cavities were filled immediately after their preparation.

(19) Yes.

(20) Yes.

(21) Yes, absolutely.

(22) In such a case, the dentine is certainly more open to infection, but the dentine of nicely cleaned cavities is rarely free from infection, and I should imagine that it would take considerable time for additional infection to do much damage to the dentine. However, I think it better to keep all cavities closed and also to sterilize the dentine before filling.

(23) I assume all this is done at one sitting; otherwise it would be foolish. It seems to me that a cavity is infected when it comes to us and is still infected when prepared. What if a little more from free saliva gets in? And what is the bacterial content of free saliva anyway? A cavity germicide may be used after drying for filling, or the thymol in thymolized cement may be depended upon, or iodoformagen

in the deep cavities, or Howe's silver nitrate, etc. How do we know that our amalgam fillings, if good ones, are not germicidal? Why was it possible to place so many gold fillings, non-cohesive and cohesive, in moderately deep cavities (not deep enough to allow much thermal shock) in the past fifty years, with so much success in avoiding pulp death and without a thought of cavity sterility? How do we know that bacteria are not starved to death by inclusion between tight fillings and live dentine, or killed by the dentinal fibrils. In fact, there is some evidence, from Hartzell's statement, that, in cases of moderate pyorrhea, there are bacteria in the pulp of (I think) thirty per cent of the pyorrhetic teeth. So far as I can determine, *all* the pulps recover from hyperemia when the pyorrhea is treated. Ergo, why should fibrils not have germicidal power?

(25) Yes. I have made no sections for examination.

(26) Very probably. It is not good practice.

(28) Caries continues, which means a species of infection.

(29) "Some time" is too unscientific and indefinite. A year is a short time for a man (some men) while an hour may be, for all I know, the full span of life for some microörganisms. (No statistics.)

(30) It may be.

(31) Yes. All clinical and bacteriological evidence supports this deduction.

(32) I do not think dentine is infected before a *considerable* time elapses. Saliva in a prepared cavity, for say a month or two, would not be serious and its effect could be removed with warm alcohol.

(33) I doubt if prepared cavities are infected by leaving them open to the saliva; not if the dentinal tubules are filled with vital protoplasm. Of course, there would be some superficial infection. However, I do not consider it good practice to leave cavities so exposed.

(35) Why should not bacteria enter dentinal tubuli, and develop there, in the absence of polymorphonuclear leukocytes to destroy them?

(36) Yes.

(37) I doubt whether an infection occurs from saliva. In the lower animals, we are certain, saliva applied to wounds is not injurious.

(38) Yes, dentine exposed to the saliva even for a short time becomes infected.

(39) There is undoubtedly a (surgically) septic surface on all dentine exposed to saliva. That the bacteria penetrate the dentinal tubuli and thus to the pulp, and cause disease in such a case as this, is believed to be very doubtful. The injury in any case is usually one that the pulp is capable of resisting. The exposure of dentine induces caries; but, if the interval is of short duration, caries is excluded as a possibility. It is "sloppy" and "bad" surgery to allow such a condition to continue; it is reprehensible. If a cavity is to be filled at a subsequent appointment, it should be varnished; and, while the varnish is still "tacky," a "high-heat" gutta-percha filling would obviate such a possibility.

(40) I think that such infection occurs more frequently than we believe. In pathological examinations of sections of teeth we find bacteria in the dentinal canals, and in the otherwise normal-appearing dentine, beyond the range of decay.

(41) Every precaution should be taken to prevent infection of a cavity during the process of cavity preparation and, if humanly possible, saliva should never be permitted to enter a cavity while preparation is being made for a filling.

(42) Anything exposed to the saliva in the mouth will, to a certain extent, become infected. Dentine, under the above condition, is infected; but not to such an extent that it could not be rendered sufficiently sterile for a safe filling operation.

(45) From a study of the histological structure of dentine, I believe infection of the dentine takes place in conditions such as those mentioned in the question.

XLIII. *May a non-vital tooth be free from infection?*

(1) Yes.

(2) Yes.

(3) Very doubtful.

(4) I have known of many non-vital teeth with and without perfect root fillings, and some without any filling (root or miscellaneous), on which, if infection existed, there had not been sufficient dissolution of membranes at the apex to be revealed by the x-ray; and where no outward systemic effect was perceptible. Under the circumstances, I would consider such cases free from infection.

(5) I believe it is impossible to find a non-vital tooth that is free from infection.

(8) A definite answer to this question cannot be made. Many such teeth may be so treated that their contents are non-infective to the surrounding tissues.

(9) It may be possible, but is not probable.

(10) Yes, clinically and to bacteriological tests.

(11) Most assuredly. There is more danger that exposed dentine will become infected, and that this infection will reach the vital pulp while the osmotic current is towards the pulp, than it will after the pulp has been removed and the canal is filled, because that current then ceases.

(12) I think I have seen such, but have not tested the matter.

(13) If by "non-vital tooth" you mean a pulpless tooth, I would say such a condition is possible.

(14) Yes.

(16) Yes. I have many sections of non-vital teeth that show no pathological conditions of periapical tissue.

(17) If a tooth has been devitalized and the root canals properly sealed, it should be considered non-infectious, unless the radiogram presents evidence to the contrary. Likewise, a tooth which has undergone a slow death in response to traumatic injury is in most cases non-infectious, and the radiographic shadows should determine the treatment to be given. However, such a tooth should be entered under strict aseptic precautions, and the canal thoroughly cleansed and sealed.

(18) It may for a short time. However, it is never safe to rely on that possibility as a preparation for root filling. Even when such teeth are making no disturbance, they are very apt to be infected.

(19) Yes.

(21) Yes, under a long chain of perfect conditions of treatment, root filling and final restoration, provided no portion of the cervix of the tooth is exposed above the enamel line.

(22) Yes.

(23) I believe one was caught once; but, whether a given non-vital tooth is free from infection, whether well treated or not, is beyond my power to tell, unless an apical shadow proves the contrary.

(24) Yes.

(25) Yes, though it is likely to become so later, by way of the blood stream or contiguous tissue.

(26) I truly believe so.

(28) Yes.

(29) I cannot say, being a *mere dentist.* (No statistics.)

(30) Yes.

(31) Yes, after treatment. I doubt it before treatment.

(32) I think so, but may be "dead wrong" clinically. I am "*dead sure*" it may be free from any infection that would affect perfect body metabolism.

(33) I think a pulpless tooth *may* be free from infection, but I believe it highly improbable that any of them are.

(35) A pulpless tooth may be very vital in its investing tissues, which may take good care of a mild infection from it; and a pulpless root germicidally, apically filled, may be absolutely free from infection. A "non-vital" (dead) tooth is one without a pulp or without life in the investing tissues, and could not be otherwise than infected, because it could have no resistance to infection.

(36) Yes.

(37) It may or may not; but the interesting question is: Is it capable of infecting other parts of the body?

(38) Yes, but it is more liable than a vital tooth to become infected.

(39) Yes, a tooth which has lost its pulp may be free from infection. The term "non-vital" is a bad term, and not in accord with the facts. Non-vital signifies *no life.* A tooth which has no life is *dead.* The pericementum may be alive, and the cellular cementum as well as the non-cellular parietal cementum may be alive. The dentine need not necessarily be necrotic, even if a tooth has lost its pulp organ. A strictly non-vital tooth is a dead or necrotic tooth—one that has lost all tissue life. Such a tooth is a foreign body in the organism.

(40) Non-vital teeth may be free from infection, but in most cases represent organs of lowered resistance which, at some future time, may become infected. Non-vital teeth should, therefore, be roentgenographically examined from time to time.

(41) I do not like the word "non-vital,"unless you apply the term to a tooth in which both the pulp and pericementum are dead. I do not believe that a pulpless tooth may be free from infection.

(42) No. The blood stream itself is not free from infection. Vital teeth are not free from infection.

(43) Yes.

(45) Yes. If the pulp was devitalized under strict surgical asepsis, and the root canal filled by an aseptic technique, I believe the tooth is free from infection. That it will remain free from infection permanently is another proposition.

XLIV. *May a vital tooth have an infected periodontal membrane (aside from that of a pyorrheal condition)?*

(1) I do not think so.

(2) I think it might, if the pulp is infected.

(3) Yes; through extension from an adjacent abscessed tooth, and on a root of a vital tooth extending into a cyst.

(4) Yes. A pericemental abscess may be due to lowered resistance of the periodontal membrane from a systemic complaint, a mechanical injury that causes local necrosis of some part of the pericementum, or a deposit of lime from the blood. It may become infected from a neighboring tooth that carries infection.

(5) I have not taken the trouble so far to find out whether a vital tooth may have an infected periodontal membrane (aside from that of a pyorrheal condition). I have found, however, that infection may extend to the pericementum of an adjoining vital tooth. While I have never examined the periodontal membrane of such an adjoining tooth, histologically nor bacteriologically, still I believe that, if the region itself was infected, the pericementum of the adjoining tooth that was encroached upon must necessarily have been infected also.

(8) Yes, possibly, in cases of pericemental abscess and similar infections; although it is not quite clear, at present, whether or not all such infections ever occur except by way of the subgingival route.

(9) I think not.

(10) Where the pulp is entirely vital and there is no pyorrhea, I have not been able to get a growth from root-end cultures after extraction; but in exposed and infected vital-pulp conditions, I often see radiographic manifestations that resemble the beginning of periapical disease. I have not cultured such root ends, and it is possible that hyperemic changes, rather than infection per se, may be the cause.

(11) Yes, the same as any other tissue of the body. Cultures may be grown at various times from almost any organ in the body. If this were not the case, we would probably not have any polymorphonuclear cells in the blood.

(12) I believe not, in the true sense. Of course, microörganisms can be found in nearly all healthy organs.

(13) Yes, there may be periodontal infection from the blood stream (which I think is very rare, however); also extension of infection from an adjacent infected area.

(14) Yes.

(16) If it may, I am inclined to the opinion that such a tooth has more than one canal and the pulp is dead in at least one of the canals.

(17) Very rarely we see a vital tooth with periodontal tissues apparently healthy and intact but the radiogram of which presents distinct evidence of periapical disease. It is probable that, in such cases, the organisms gained entrance at the gingiva and travelled through the periodontal membrane without producing macroscopic injury to this membrane. Minute study of the membrane would show the path of the organisms and the resultant inflammation. In such a case, mixed infection follows after a time and eventually the tooth takes on pyorrheal characteristics. Usually there is a history of trauma in such cases.

(18) There is no reason why the periodontal membrane may not become infected just as any other tissue may, if infection can get to it, and it sometimes does; though, as a rule, infection, aside from that of a pyorrheal condition, is very rare.

(19) Yes.

(21) Yes, as in cases of trauma, poor contact points, malocclusion due to improperly contoured occlusal surfaces along bridges, etc.

(22) Yes, tissues often contain infection, but infection does not always mean disease.

(23) Price says that when a pulp is exposed by decay (at the bone), the apical tissue may then have bacteria in it. I think I quote him correctly (Price's lecture, in 1917, before state societies). It is theoretically possible that a blow might produce a non-septic pericementitis (vital pulp) and then this might be infected by way of the circulation. Clinically, I have never seen anything that looked like this. Theoretically and clinically, an abscess on one tooth may spread

to the pericementum of the next vital tooth. The infection (pericementitis) may or may not kill the pulp of the second tooth. Sometimes, teeth adjoining abscessed teeth have had to be extracted for this reason.

(24) Yes, but it is the exception.

(25) Yes, phagedenic pericementitis. I have a case now that seems to be neither the above nor pyorrhea, but a local necrosis, the cause for which I cannot locate, unless it be an adjacent extraction four years ago.

(26) I cannot see why it would not. Traumatic occlusion and a thickening of the membrane might follow from the action of organisms carried through the blood stream, or even through interproximal spaces to the membrane.

(28) No; or, if so, only a partial infection.

(29) See my answer to question XLIII. (No statistics.)

(30) Yes.

(31) Yes, from a metastatic abscess or from the blood stream following trauma.

(32) It certainly may. I have one patient who has hypercementosis on nearly all molars and bicuspids, and they are sound and vital. He is a grain man and daily tests the quality of wheat by grinding the grains in his mouth.

(33) Yes, a vital tooth may have an infected periodontal membrane and it may be benignly infected (distinct from pathological infection). I would not expect this infection to reach very far toward the apex. I do not consider that it has any particular clinical or pathologic significance.

(35) In the presence of low resistance, infection may be carried from another focus and develop in the apical region of a tooth with a vital pulp.

(37) Yes. A traumatic injury may result, e.g., a fish-bone or tooth-pick accident.

(38) There is a possibility that the periodontal membrane of a vital tooth may be infected, for example, from trauma; but it is sufficiently rare to be disregarded.

(39) The pericementum may become infected and the tooth may retain the health of its pulp, or vice versa. The presence of pus, as in

suppurating pericementoclasia ("pyorrhea alveolaris"), does not preclude the presence of a healthy pulp; but a suppurating pulp does preclude the presence of a healthy periapical pericementum. It is conceivable that the pericementum of a tooth may become partially destroyed through an infection in a localized area on the cervical surface of the root, and that this infection may not extend over nor include the entire periodontal structure. The pericementum may be infected and ulcerative in a minute area, and this lesion may constitute the entire diseased area. The periapical area (it is believed) may become infected previous to the parietal area, and the vitality of the pulp may be endangered through an extension of infection; yet, from the facts, the pulp could be considered vital but not healthy. Pyorrhea is a sequel of pericemental infection and pus is its distinguishing symptom. Periodontoclasia endangers the pulp by an extension of its disease processes.

(40) If the word vital means that the sensation of pain is present (for example, when an instrument is inserted into the pulp), I should say that such a condition is very frequent. If a pulp has become infected, the nerve fibres are not immediately destroyed but may remain vital even after the disease has progressed to the periodontal tissues.

(41) I think it is quite possible for the periodontal membrane to become infected from other sources than from a pyorrheal condition.

(42) I can conceive that this might happen, but very rarely to the extent of producing a pathological state.

(45) Yes. It may be vital and still have an abscess of the periodontal membrane. Black speaks of the condition as a lateral abscess of the pericementum.

(?) Yes.

XLV. *Does an infection at a root end come (a) from the tooth itself (b) through the canal (c) by the presence of infected dentine; or (d) is it carried there from the blood stream independently of infection in the canal? In case you consider that an apical infection comes from the tooth itself or the canal, how do you account for (e) the presence of infection in canals and at apices that have undamaged natural crowns? (f) If you consider that such an infection comes from the blood stream, why is it that, in a given mouth, some non-vital teeth are not infected and others, adjacent, may be badly infected?*

(1) From my experience, I think it comes from infected pulps. Some pulps die from severe shock or blows, severe strains, and sometimes from severe thermal shock, causing congestion and strangulation of pulp. I don't believe a pulp ever dies from blood-stream infection alone; but, if it has had such shocks or blows as those mentioned above, it is rendered liable to blood-stream infection.

(2) (a) No. (b) Yes. (c) Yes. (d) I think it might be, in certain cases of lowered resistance. (e) If the canals *are* infected, I think the infection might have come through a pyorrheal condition. (f) The supporting tissues of some teeth may have a lowered resistance.

(3) Infection at a root end comes from the canal, but may also come alongside of the root in cases of pyorrhea. A tooth with an undamaged natural crown may have a non-vital pulp, caused by accident, etc. Any decomposition of the pulp may produce apical involvement.

(4) My belief is that most infections at the root end are carried through the apical foramen by accumulation and expansion of gases within the tooth, due to decomposition of the pulp. It could not come from the tooth itself, unless the tooth had become infected—that is, through the dentinal tubuli. We seem to have it too well fixed in our minds that either dry or moist gangrene of a pulp is limited to the apex. It would be a beautiful provision of nature if it were limited to the apex, but I think there may be death and decomposition of the nerves and blood vessels beyond the apical foramen, in which case nature must rebel. I believe that infection is carried in the blood stream to the apices of teeth with large and open foramina, and where the contents of the canals are putrescent. In answer to question (f): I believe that some non-vital teeth show no apical infection, due to the closure of the foramen by a non-septic particle, which prevents contamination from the infectious pulp contents. There may be apical infection in the same mouth when such closure does not occur.

(5) An infection at the root end comes most frequently from the tooth itself "through the canal, by the presence of infected dentine," but I believe it may also arise from the blood stream, devitalization of the tooth having lowered the resistance of the apical and periapical tissues. The presence of infection in canals and apices of teeth with undamaged natural crowns, is due to the fact that the pulp tissue of such a tooth, devitalized by a trauma, is a locus minoris resistentiae.

Where the question (e) refers to non-infected teeth and adjoining badly infected ones, I hope such conclusions are not arrived at through radiographic findings. If, however, such findings are the outcome of bacteriological examination, they do not mean much, because the present technic for such examinations is far from being perfect. One must consider, however, each individual tooth, whether and how it has been treated, and to what extent the natural resistance to infection at the apex may have been further lowered by trauma and other conditions.

(8) My opinion is that seldom, if ever, are the periapical or pulpal tissues infected by blood-borne organisms, except as they infiltrate from the gingival areas of the tooth affected, or of teeth adjacent to it.

(9) Infection comes most frequently "through the canal," from infected pulp or dentine. Blood-stream infection does occur; there may be also extensions from infected gingivae or pyorrheal pockets.

(10) Root-end infection obviously comes through the canal in most instances. The proof of this is seen in the fact that, until the pulp becomes infected or loses its vitality, the periapical tissues remain normal; and in the further fact that operative procedure in the canal is so frequently followed by periapical symptoms. Contamination by infected dentine must travel by way of the canal to infect the root end. It is probable that much infection finds its way to the apical region by means of the lymph channels of the pericementum, being carried there from the gingival crevice. This is probably a frequent source of infection in teeth with undamaged natural crowns, in which the pulp has lost its vitality through traumatic shock. Aside from such traumatic cases, I have never seen periapical destruction or pulp death in a tooth with an intact enamel covering, unless it was continuous with a region of periapical destruction about the apex of an adjoining pulpless tooth. Many teeth with pulps that were devitalized by traumatic shock, in which the enamel covering is unbroken, later develop periapical cysts. I have never been able to get bacterial growths from such cases. I think it is more possible than probable that infection carried by the blood stream finds lodgment at the apex of a normal vital tooth. If such a premise can be proven in any given case, I cannot see what good treatment or extraction would do, until the source of such infection was discovered and eliminated.

(11) (a) It may. (b) It may. (c) It may. (d) It may, provided there is a proper culture medium near the apex of the tooth. (e) Derived from infection traveling in the blood stream. (f) Because of greater resistance at this particular part, or greater accessibility for anti-bodies.

(13) (a) Undoubtedly through the root canal in a vast majority of cases. (d) Occasionally, probably, through the blood stream. (e) Through the lymphatic glands of the periodontal membrane or pyorrhea pockets, and through the blood stream, accompanied by interference with the circulation in the pulp. (f) In a case where the infection comes from the blood stream, unfavorable conditions in the apical portion of the canal of an infected pulp undoubtedly contribute to the result.

(14) I believe infection "comes" both ways. Some non-vital teeth show no infection in the apical region because nature has so walled off the dentine of the tooth that there is practically no connection between it and the surrounding tissue. Where nature has not done this, the apical region may become infected from the tooth, through the root canal or from the blood stream.

(15) Probably either may be a source of infection. Some pulpless teeth in the same jaw become infected while others do not because of the destruction of the apical periodontal membrane by previous serious infections, by over-medication with caustic remedies, or by arsenic used in the destruction of the pulps, any of which may cause a lowered resistance.

(16) Where infection occurs in teeth without caries, some influence has caused a lowered resistance in the pulp—the bacteria carried to the pulp through the blood stream caused the pulp to die, resulting in an irritation of the periapical tissue and infection. I believe that such non-vital teeth do not invariably become infected because the bacteria differ in infective quality, just as the resistance of the periapical tissue varies.

(17) In a majority of cases, the infection comes from the tooth itself, through its canal, either because of careless root-canal technic, or neglected purulent pulp. Rarely infectious material is carried in the blood stream to the locus minoris resistentiae created by devitalization of the pulp. The rare cases of apical infection about teeth with

undamaged natural crowns can be explained by the theory that the organisms gain entrance at the gingiva, following some slight trauma, and travel through the periodontal membrane to the apical space, without producing any apparent changes in the membrane at the beginning. Trauma of malocclusion accounts for the blood-borne infection that involves some non-vital teeth, while adjacent ones are left healthful. The teeth that become infected are those that are doing more than their share of work because of shifting, reconstructions, loss of teeth, etc.

(18) Root-end infections occur both "through the canal and by the presence of infected dentine," and are carried there through the blood stream, independently of infection in the canal. A majority, however, occur as a result of infection through the canal. Infection occurs at the apices of some teeth, and fails to occur at the apices of other teeth, because of their selective qualities; or, in other words, bacteria locate where they find material on which they can propagate, and infection does not occur in other teeth because that condition is not found by the organism itself.

(19) We believe that in nearly all cases the infection comes from the canal. The presence of infected dentine, we believe, is a contributory factor. We think it possible, but *very* improbable, that infection takes place by way of the blood stream, independently of infection in the canal. We believe that, in cases of infection at apices with undamaged natural crowns, the infections have reached the root-ends by way of damaged periodontal membranes, or its lymphatics. We believe that prevention of infection at the root ends of non-vital teeth depends on two factors, namely, resistance of the tissues at that particular point, and resistance of the body as a whole.

(21) (a) The tooth itself, yes. (b) Yes. (c) Yes. (d) If the patient is in very poor physical condition—resistance low—it is possible. (e) Infection can easily pass through cementum above the enamel line, if there is any absorption of alveolus and recession of gum. (f) The size of the canals, and whether straight or tortuous, influence this condition; so does the amount of recession around the necks of different teeth.

(22) Infection at a root-end may appear in any of the ways mentioned. (d) It is probably brought by the blood stream. (f) A

difference in the tissue condition. In my opinion, the infection is secondary, i.e., does not occur in sound tissue, but only in regions of lowered resistance.

(23) (a) I believe a tooth itself is aseptic. (b) From the infected pulp canal, bacteria traveling to the apical tissue. (c) See my answers to questions XXXVII and XL. (d) This is possible when another focus is present, as, for example, appendicitis, another apical abscess, a boil, etc.; otherwise, this is a mere theory. We need not look far from the tooth to find opportunity for infection. (e) All teeth have porosities capable of admitting infection. Exposure of the root at the neck explains it. This process would take time. Clinical evidence shows that formation of abscesses about teeth killed by blows, etc., is delayed. (Statistics on the length of time after death of teeth that granulomatous conditions first appear are not available.) (f) Dr. Best evidently agrees with my view that resistance of blood infection is secondary to that of *infection from the canal* (see my answers to questions XV and XL).

(24) Infection comes through the root canal in a great majority of cases. It may exceptionally be carried there from the blood stream. The crown of the affected tooth may be only seemingly undamaged. There may be microscopic openings in it through which bacteria have gained access to the pulp chamber.

(25) My opinion is that the infection comes, in a majority of cases, from the canal, but may also come by way of the blood stream, if local tissue resistance is lowered from any cause. However, there are teeth, the dentine of which is saturated with toxic substances, which are condemned because of a general periodontal disturbance, and not merely because of infection at the foramen. (e) Due to blood-borne infection, which found lodgment in an area of lowered local-tissue resistance. (f) In recent years, when asepsis was not considered, much infection was carried to the apical tissues in attempting pulp removal. Therefore, the tooth in which the more careful effort at root operation was made was the more likely to suffer. Over-drugging has also destroyed tissue and lessened resistance.

(26) (a) Possibly. (b) Yes. (c) Yes. (d) Either way. (f) Traumatic occlusion must always be considered in such cases.

(28) (a) Usually. (b) Yes. (c) No. (d) No. (e) There is no *infection* in such teeth. The pulp in such a tooth may be dead, but there is no infection.

(29) My answers to questions XLIII and XLIV apply here, also, only more so. (No statistics.)

(30) Infection at a root end may come from the tooth itself, or from the blood stream, or lymph channels. I do not know why some teeth are infected by the blood stream, and some are not, except that resistance of the tissues about the root ends varies; and those that are less resistant naturally are the first to be infected.

(31) I believe it may come from either source. Regions of lowered resistance may be responsible in either case, in my opinion, for the selective localization of infection.

(32) I have not yet observed a case where the infection did not come from the canal, or dentine, or as a consequence of trauma.

(33) I believe that most periapical infection comes through the canal. I do not dispute the possibility of hematic periapical infection, but I doubt if it occurs very often. When the natural crown is undamaged, I believe the infection comes from the blood. In the case of an abscessed tooth, with an undamaged natural crown, there is always in the canal a great amount of disintegrating pabulum for bacteria. On the other hand, if a pulpless tooth has been treated by a dentist, most of the pulp (at least) has usually been removed. *In a general way*, the larger the number of bacteria, the greater the probability of abscess formation, and the more food (dead pulp) there is for the bacteria, the more likely they are to live and thrive; so that, in cases where the natural crown remains undamaged and the pulp dies, there is much bacterial growth, and practically always an abscess. In teeth where most or all of the pulp has been removed, the amount of bacterial growth (and so the probability of abscess formation) varies, depending on the amount of pabulum and the amount of infection introduced from the mouth through the canal.

(35) The most frequent and usual cause of apical infection is "through the canal." Sometimes, not often, the cause may be "the presence of infected dentine." Conveyance from the blood stream is very seldom the cause—if there is apical infection and a vital pulp, it may be the cause. This is very rare, unless there is intense infec-

tion nearby. Of course, there may be a combination of causes, though this is still less frequent in occurrence. (e) A blow, a strong jar, or occlusal trauma, sometimes causes pulp death under sound dentine and enamel. (f) Infection carried by the blood stream is an unusual cause for an apical abscess. Usually, such an abscess is caused by bacteria from the root canal of a pulpless tooth.

(36) (b) Usually through the canal. (c) Often through infected dentine. (d) I have never seen this theory substantiated clinically. (f) Differences in general and local resistance of the tissues may account for such a phenomenon.

(37) I am unable to answer.

(38) Infection may come from canal contents, infected dentine, lymphatic circulation, and possibly from the blood stream. Local conditions, especially canal operations, and injury to periapical tissues, explain why some pulpless teeth become infected and others do not.

(40) The infection, in most cases, comes from the tooth. In a number of cases of which I have roentgenograms, the infection started from another around a neighboring tooth. I do not believe infection will be carried there, through the blood stream, unless the tissue at the apex of the tooth has a lowered resistance.

(42) (a) The tooth itself. (b) Through the canal. (c) The dentine would be infected in a case of putrescent pulp. (d) *So rarely as to be negligible.* (e) In a case of infected canals and undamaged natural crown, where apical infection supervenes: it may be caused by a blow; by sudden changes of temperature, especially of heat; or by some agency forcing infection from the canals through the apices of the roots.

(45) (a, b, c) Sometimes infection of a root end comes from infected dentine involving the pulp, which gradually spreads to the apical region. I thoroughly believe that some cases of root-end infection arise from septic root-canal work. (d) Some root-end infections, I believe, are hematogenous. I cannot explain their occurrence in any other way. Ulrich believes that blood-borne infection is the explanation for the majority of apical abscesses. (f) Differences in the local resistance of the apical region would explain why not all non-vital teeth are infected when the positive infections are hematogenous. Then, too, the non-infectious teeth may have been infected and may

have undergone resolution, which may "light up" again if the opsonic index of the individual is suddenly lowered. It is a well known fact that the contents of some dental granulomata are sterile. It seems to me that well known laws of resistance and immunity explain why some non-vital teeth are not infected by organisms of hematogenous origin that infect adjacent teeth.

XLVI. *Do you believe that the presence of silicate fillings causes pulp death? If so, how do you prevent such action?*

(1) I always place a little varnish in the bottom of a deep seated cavity, if it is near the nerve, before inserting the filling. All kinds of fillings inserted very near vital pulps are dangerous, if one does not take the precaution to line the bottom of each such cavity with either varnish or oil of cloves mixed with cement powder, as given in previous answers.

(2) If placed too near a pulp, yes. Prevent pulp death with zinc cements or insulating varnishes over the pulp.

(3) I have never used such a filling close to a pulp. I have not had any pulp deaths from the use of silicate fillings. I do not know the composition of the material; therefore, I am not in a position to judge.

(4) I believe that any filling, silicate, the phosphates, all cements, can cause the death of pulps. I have not had a pulp death under a silicate filling of four years' standing. For all deep cavities I use an intervening substance, usually Caulk's cavity lining.

(8) Yes. Protective cavity linings prevent pulp death.

(9) Yes, in some cases, if close to the pulp. Use an insolation of resin in ether to prevent pulp death.

(10) No. We have had no pulp deaths under silicate fillings and only one under a gold inlay in the past four years. The routine use of the treatment outlined in my answer to question XLII has convinced me that pulps die under fillings from only two causes: infection and traumatic injury.

(11) The dentine under silicate fillings should be protected with a non-conductor.

(12) Possibly; an oxyphosphate lining beneath a deep filling is a good precaution.

(13) No, not more so than that of any other class of fillings. A large proportion of the pulps that die under cement fillings of any kind, die through previous infection, I believe, rather than from an action of the cement filling. I prevent pulp-death by using the best judgment I have and leaving no infected (decalcified) dentine over the pulp. Where I fear thermal shock, I use non-conducting cavity lining over the pulpal or axial wall before introducing cement or any other filling material.

(14) Yes. Precautions: use care in mixing the filling. Line the cavity with cavity varnish; and, if it is deep, use a cement lining.

(16) My experience leads me to believe it sometimes does do so. I cannot prevent such action.

(17) Yes, silicate fillings cause pulp death. We should guard against such an eventuality by using a good intermediate layer as a pulp protector.

(18) There can be no doubt that pulps die from the action of poisonous material in silicate fillings. The problem of how to prevent it is a difficult one. Tinfoil, shellac, or varnish, laid over the pulp, sometimes gives assurance of protection. However, I believe that after a time silicate fillings lose some of their devitalization properties; and that, if the pulp itself can withstand the strain for a certain period of time, the irritation is relieved accordingly.

(19) Yes. Attempt to prevent it by lining the cavity.

(20) I have seen teeth where pulp death was caused, I believe, by Ascher's Artificial Enamel. When a silicate filling is used in a deep cavity, a zinc oxyphosphate lining should be employed.

(21) Pulp death does not occur unless teeth are filled very close to the pulp. Possibly silicate fillings have the same approximate effect in this regard as those of metal. Deep seated cavities should be lined with an agent that prevents penetration of the tubuli by free acid from the mix of silicate.

(22) I think that a chemico-physical compound like a cement may cause pulp death, if it is placed too near the pulp. Use a varnish of chloroform and pure rubber.

(23) I do not think so. I have to remove such fillings occasionally and almost always find the teeth vital; if non-vital, they might have been so originally. For very deep cavities I think a layer of "cavi-

ticide," or an antiseptic varnish—almost any—plus hydronaphthol, a sufficient intermediate. Sometimes Iodoformagen and thymolized cement are introduced for good reasons, and silicate over them. See my answer to question V.

(25) I have observed more pulp deaths following use of these fillings than of any others, but leaking has been a frequent factor in this relation. The cavity floor should be disinfected with thymol, then varnished, and coated with oxyphosphate-of-zinc cement.

(26) Not more frequently than from the use of other fillings.

(28) *Yes.* It may be prevented *by using an intermediary* of varnish or cement.

(29) The insertion of a silicate filling over a *nearly exposed pulp* undoubtedly causes its death. Such results are prevented by not inserting such fillings. (No statistics.)

(31) In my experiemce the liquid (acid) in silicate-cement mixtures is not so thoroughly neutralized by the powder as it is in zinc-phosphate cements; and, if it is placed in too close proximity to the pulp, it will decalcify the remaining dentine and produce death of the pulp by chemical irritation. A suitable lining—insoluble, or nearly so, in the acid—will prevent this.

(32) I protect with a cavity lining.

(33) I have found so many dead pulps under silicate fillings that I am inclined to believe that some of the silicate materials, of the past at least, exerted destructive action on pulps. Such action may be prevented by the use of intermediates of oxyphosphate-of-zinc cement and of cavity linings such as chloro-rosin.

(35) Probably. Silicate cements carry the blame belonging to infected dentine left beneath them.

(36) Silicate-cement fillings, in years past, in my experience, have caused the death of teeth. These fillings were in both shallow and deep cavities. I have not noticed that this occurred to any great extent in recent years. I have used silicate cement very seldom and then only in shallow cavities. I have used and do use porcelain inlays extensively, and do not notice the same destructive influence.

(37) The *early* silicates caused death of pulp tissue. Present-day silicates do not seem to do so.

(38) The greater frequency of pulp death under silicate fillings than under metallic restorations indicates that silicates have been an important factor in devitalization. The injurious effect of silicates upon the pulp may be prevented by more care in cavity preparation and by the use of protective media on the cavity walls.

(40) I have seen a large number of roentgenograms showing periapical infection due to pulp death from silicate fillings. I think it would be interesting to classify these cases according to the brand of the material used.

(41) Up to the present time I have seen no evidence that silicate fillings caused death of pulps, where, under similar circumstances, the pulps would not have died under fillings of other materials.

(42) Yes, in some cases; not in all by any means. Pulp death may be prevented by the use of cavity varnish; also by avoidance of too great pressure with the celluloid strip in placing the filling. The pulps of lateral incisors, especially, are endangered by the incautious use of condensing pressure on the strip.

(45) Yes. It may be due to free acid that arises when the silicates change from a plastic to a crystalline form. I always use a balsam varnish to seal the tubuli of the tooth before a silicate filling is inserted.

XLVII. *If you take the stand that all pulpless teeth, regardless of radiographic findings and regardless of the patient's health (good or poor), should be extracted, do you put any age limit on such procedure? Why?*

(1) I do not take such a stand.

(2) I do not believe in such wholesale mutilation of dental organs.

(3) I have not as yet been convinced that all pulpless teeth should be removed.

(4) I do not take such a stand; but, should I, neither age nor health would bar the procedure, except, of course, in case of a very old, very sick, and very feeble person, and where there were many non-vital teeth. I think age is not a factor to be considered, if the circulatory system is not impaired.

(5) I do not take the stand that all pulpless teeth, regardless of radiographic findings, and regardless of the patient's health (good or poor), should be extracted.

(9) I don't take such a stand. I think it absolutely idiotic.

(10) I antagonize such a stand.

(11) I do not take the stand that all pulpless teeth should be extracted. (I see that my questioner has reformed and is using the correct term, "pulpless teeth." Congratulations!)

(12) I don't take the stand.

(13) I do not take such a stand; but, if I did, I think I would limit such indiscriminate and radical action to patients under six months of age and to those who are over one hundred years old.

(14) I do not take this stand.

(15) I do not take the stand that all pulpless teeth should be removed under such circumstances.

(17) Radiographic findings should be the deciding factor in determining retention or extraction of pulpless teeth, especially such as have canal fillings. The health and age of the patient should be considered only in regard to physical fitness to withstand the operation of removal.

(18) I do not take the stand that all pulpless teeth should be extracted regardless of the radiographic findings, if the patient is in good health.

(19) We do not take this stand. If we did, it would apply to patients past middle life, as their resistance to infection is lower.

(21) I do not take such a stand.

(22) I do not take any such stand.

(23) No age limit. See my answer to question XVII.

(24) I do not take this stand at all. I believe it is pernicious teaching.

(25) I do not take such a stand.

(26) Such a stand will never get us anywhere, and should not be countenanced by any one who calls himself a dentist.

(28) I do not take such a stand.

(29) I do not take such a "fool" stand. (No statistics.)

(30) I do not take such a stand.

(31) I do not take this stand.

(32) I don't take such a stand, for it seems unscientific in the light of careful clinical observation.

(33) I have *never* taken the stand that all pulpless teeth, regardless of radiographic findings, and regardless of the patient's health (good or poor), should be extracted. I am opposed to such a stand; yet I never lose sight of the fact that the only satisfactory solution of the whole pulpless-tooth problem is prevention of death of dental pulps for the coming generation. There is no absolutely satisfactory solution of the pulpless-tooth problem for the adults of this generation.

(35) Only such pulpless teeth should be extracted as radiographically show infection, or may, from the appearance of surrounding conditions, be justly suspected of causing it. It is not the number of years, but the amount or lack of systemic resistance, that should influence our judgment. A mild infection in the presence of high resistance may be expected to be destroyed in time, provided proper successful local work is done so that danger will be averted in case resistance should be lowered in the distant future.

(36) With the patient at thirty years, the trouble from the altered conditions increases as age develops, with the exception that in some individuals the influence of acquired immunity comes in, as does also a peculiar improvement in vital resistance, brought about by altered habits and other subtle influences.

(37) I do not take this stand.

(38) This position is not warranted by present knowledge.

(39) I take no such stand. Why pick on a healthy tooth because he is old?

(40) I do not take the stand that all pulpless teeth should be extracted.

(42) I am opposed to any such stand by intelligent, thinking, men.

(45) I have not as yet taken the stand that all non-vital teeth should be extracted.

XLVIII. *Do you consider that the manner of handling hospital cases, relative to problems of pulpless teeth, should govern the management of patients in private practice?*

(1) Yes, unless the hospital has a well equipped clinic and an efficient dental surgeon belonging to the hospital staff.

(2) No.

(3) I do not, for the average case in private practice differs greatly from the hospital case—where the patient's resistance has been greatly

reduced, he is suffering from one or many ailments, and quite often neither time nor conditions permit extended operations upon the teeth.

(4) The facilities for treating hospital cases in our city are such that, even with the inclination to do so, the possibility would be precluded of giving a patient the same treatment that should and would be accorded at our office. I think we are forced to do many things at a hospital that are contrary to our wishes, and differently from the way we manage cases in a private office.

(8) No.

(9) No.

(10) Good judgment is necessary in all cases, and conditions often govern treatment.

(11) I do not understand your question. I believe the laws of disease or health are the same in a hospital, private office, and college clinic. Personally, I would like to have the money consideration entirely "*extracted*" from this great question.

(12) No; hospital cases must be handled in a more radical way.

(13) No; conditions are very different.

(14) No.

(15) Yes; but to accomplish this, dental internships are a necessity.

(16) Yes; where systemic conditions are present.

(17) No. Patients who are able to go to a private office can be handled with more freedom than those who are confined in a hospital. A patient in a hospital is more or less seriously sick, and should be given careful and considerate attention.

(18) The same consideration should be given to patients in private practice as to those in hospitals, so far as it is possible to do so. The nearer we can approach the ideal in the management of all cases, the better.

(19) No; on account of differences in facilities, the patient's condition, etc.

(20) No. In a general way, hospital cases are sick cases. Patients in private practice are usually healthy. The follow-up system for hospital cases is difficult or impossible to execute. In private practice we can and do see our patients at frequent intervals, and keep informed regarding their physical conditions.

(21) No. Hospital cases are usually affected by other than dental conditions—are complex. Patients in private practice that fall in the same category should receive the sort of treatment that is given to hospital cases; otherwise it is not necessary.

(22) I do not believe that a patient is in a hospital because of his pulpless teeth. Neither in hospital cases nor in those of private practice should I remove all pulpless teeth, but only those that in my opinion were beyond saving.

(23) This raises the question of right and wrong method; also the control, by patients, of the disposition of their own teeth. Dr. Garretson used to say that Dr. Morell Mackenzie, in treating the Emperor Frederick's throat, "made the mistake of thinking of him as different from a beggar." (See my answer to question III for comment on the ethics of the situation.)

(24) Yes.

(25) No. Patients in private practice usually are in better health than hospital cases, and office facilities for the care of private patients are better than those at a hospital.

(26) No.

(28) You should assure yourself that the hospital is correct, or handle the case according to your own judgment.

(29) No personal experience. (No statistics.)

(30) In a general way, yes.

(31) An ambiguous question. Locality or facilities for the treatment of pulpless teeth should be governed only by the gravity of the case, the post-operative results, the recommendations of the operator, and the concurrence of the patient.

(32) *No, most emphatically;* but it is of great value.

(33) Common sense and keen consideration for the patient should govern the handling of all cases, whether hospital or ambulatory.

(35) No, because hospital practice cannot devote the time necessary to effective root-canal technique.

(36) No. Questions of time, economy, and gravity of the case, govern this matter.

(37) Yes; providing that radiographic evidence concerning the periapical region is positive.

(38) No.

(40) I do not think that the manner of handling hospital cases should govern the management of patients in private practice, because it is advisable with hospital and charity patients to be more radical, in order to save their time and prevent expense. Such patients can usually ill afford to have teeth treated when the prospects are that, after prolonged treatment, the teeth will have to be extracted.

(41) In my estimation every dental clinic should be an integral part of a general hospital. With the well-established fact that oral conditions are closely associated with systemic infection, I can see no reason for separating pathological mouth conditions from any other diseased conditions. I am convinced that all such cases would be very much better treated in a hospital, if the same care and method of procedure were carried on in its dental clinic as are observed in the other departments of a well-regulated hospital. There is no other way, of which I have any knowledge, by which dentists and dental students may obtain a correct vision of the importance of their work and its relation to the general well-being of the patient; and there is certainly no other way whereby dentists or dental students may live in the atmosphere of an institution devoted to the care, and restoration to health, of those who are ill. The environment of many dental offices—the entire lack and ignorance of any approach to surgical cleanliness— is anything but flattering to the profession of dentistry at the present time. The dentists of the future must have a vision which has been denied the dentist of the past, if he is to measure up to his full responsibility and capability. Dentistry today does not mean simply "*teeth.*"

(42) Not at all.

(45) No, I do not. In private practice the aseptic handling of a root canal is possible and the following up of the case by frequent roentgenographic examinations is possible; while for hospital cases, the financial means or the skilled root-canal operator is not always available.

XLIX. *What do you think of the extraction of pulpless teeth that radiographically show no infection, in patients in good health?*

(1) I think no skilled conscientious dentist would do it.

(2) If they are extracted for the sole reason that they are pulpless, I think it is totally unwarranted.

(3) I would fill the roots and leave the teeth in the mouth.

(4) I see no justification for the extraction of a pulpless tooth in the mouth of a patient in good health, and especially if the radiogram shows no indication of infection.

(5) There is no reason in the world why pulpless teeth in patients in good health should not first be subjected to dental treatment, if there is a fair chance of saving them. Such teeth should be removed only if and when the treatment is unsuccessful.

(8) I am opposed to such practice.

(9) Criminal.

(10) I consider it unwarranted mutilation, based upon panic.

(11) I have never been guilty of such a crime, except when ordered to do so by the patient's physician, whom I then regarded, and still regard, as grossly ignorant.

(12) Bad practice.

(13) I think the operation is unjustified, and I would perform it only upon physicians and dentists who advocate it (provided, of course, that the teeth were useful to the patient, if retained).

(14) I consider it a serious error.

(15) It is an injustice to patients. I doubt if any dentist with a well-balanced mind would have such teeth removed from his own mouth.

(16) Pulpless teeth that do not show infection radiographically should not, in my opinion, be extracted.

(17) In my opinion, it is entirely too radical. I feel that we can safely rely upon the radiographic shadows as our guide to extractions.

(18) I see no reason for removing pulpless teeth that radiographically show no infection, in a patient in good health, unless, however, that patient's family history suggests a tendency toward cardio-renal disturbance. If the patient's family history indicates that a large number in that family have fallen victims to kidney or cardiogenic diseases, in early or mature life, then I believe the extraction of such teeth as a method of *prevention* would be good practice.

(19) I do not think it is indicated.

(20) We do not recommend it.

(21) I do not approve of this practice, and do not do it.

(22) I think that the extraction of such teeth is malpractice.

(23) I think well of their extraction as a prophylactic measure. The patients frequently think otherwise, so often I don't know what to do.

(24) I think it is malpractice.

(25) If a good root-canal operation, as shown by the x-ray, may be done, extraction is not justified, because we have not yet been able to assure our patient that he will lose his health from retention of such teeth, or that his health will be maintained if such a tooth is removed.

(26) Ridiculous; unprofessional; malpractice.

(28) I would not recommend such practice.

(29) As "*radiographs*" show neither the presence nor absence of infection, the question cannot be answered. (No statistics.)

(30) I think this should be discouraged.

(31) I do not believe it is necessary or advisable.

(32) Entirely unwarranted, yet daily advised in Kansas City, hundreds of times probably.

(33) I do not believe we know enough about the problem of metastatic infection arising from a dental focus to justify such a radical procedure as extraction of all pulpless teeth, even in the mouths of patients in good health.

(35) A fad, like Emetine; sometimes an honest error, caused by not taking physiology, pathology, and bacteriology, into account.

(36) In my opinion, it is overdone.

(37) I do not think it is justified by present knowledge.

(38) It is malpractice, and indicative of an erratic mentality, to which the care of his fellow men should not be entrusted.

(40) I think they should be retained.

(42) I am opposed to it. It is about as rational as it would be for a man to have his legs cut off while he could have the operation done expertly and neatly, rather than run the risk of having them cut off in a railroad accident at a time when he might not be feeling just fit to stand it.

(45) At the present time, I am of the opinion that it is good practice to make roentgenographic examination of such teeth, about once or twice a year, and not to resort to extraction so long as the resistance of the patient stays high and the individual remains in good health.

If rarefied-bone regions develop, the teeth should then be extracted and the areas curetted.

L. *How do you view the construction of extensive restorations that call for the use of pulpless teeth as abutments?*

(1) It is all right if there is no sign of infection, and if the pulpless teeth are properly filled.

(2) If the teeth are already pulpless, but otherwise in good condition, I would use them as abutments if necessary, but I do not believe in devitalization for such a purpose.

(3) If fixed: I am less in favor of them every time I see one. If removable: I am in favor of them in many cases.

(4) Unfavorably, if it is to be a fixed appliance.

(8) I am opposed to such practice.

(9) If the teeth are already pulpless, and in good health, and the patient is in good health, I can see no reason why it should not be done. But I would not eradicate a healthy pulp to make such a restoration, for it is not necessary.

(10) In the overwhelming majority of cases, such restorations are unnecessary. It is an extremely rare occurrence in our office to devitalize pulps simply for the construction of restorations.

(11) I do not teach the attachment of large restorations to natural teeth. (I see my questioner is still converted to the term "pulpless teeth.")

(12) Viewed doubtfully.

(13) I have never been in favor of large restorations in the form of fixed bridgework. Justification decreases as size of appliance increases. With removable appliances, conditions become more favorable.

(14) I undertake a certain amount of such work, if the patient is in good health; and if he is willing to assume the responsibility for what it may be necessary to do later, should some real research worker some day prove that pulpless teeth are a menace to health.

(15) No tooth is equal to such an additional load. Sooner or later it gives way under the stress. Examine radiographs, and see the condition, of the periodontal membrane of teeth bearing this extra stress; and observe the difference between the attachment of the periodontal membrane of such teeth and of those not subject to the additional strain.

(17) In no case would I favor devitalization in order to get a bridge abutment or make a restoration of any nature. In case the tooth had already been devitalized and the root canals properly sealed, and the radiogram of the tooth were negative, it would be good dentistry to make use of the tooth as an abutment in a restoration.

(18) It is not good practice, in making large restorations, to rely on pulpless teeth as abutments. There are many striking examples in every man's practice of teeth of that type that do years of service, but which are bound, if great strain is placed upon them, sooner or later to become infected.

(19) Viewed with suspicion.

(20) I disapprove of using large restorations that require the devitalization of pulps. In general I prefer not to place large restorations with pulpless teeth as abutments.

(21) If radiograms and the case history show that such pulpless teeth are not diseased, I would use them as abutment teeth for restorations.

(22) I think that pulpless teeth are able in part to support a reasonable restoration device, but of course they must not be overloaded.

(23) My answer to question III presents my views in this relation.

(24) If the teeth are already pulpless and apparently free from infection, I believe it is justifiable to use them for abutments. I do not believe in sacrificing the pulps of vital teeth to make abutments.

(25) I have discontinued such practice, though I can conceive of individual instances as favorable, when one might be justified in supporting four teeth between two abutments.

(26) Such a case is not better than the most questionable root.

(28) At best they are only temporary.

(29) It would depend entirely upon whose "construction" they were. If of my own, I might be more charitable in their criticism than otherwise. (No statistics.)

(30) I can see no objection to this, if the abutments show no evidence of apical involvement. I do, as a rule, discourage the use of fixed bridgework, preferring a removable type of appliance.

(31) Viewed with ill favor. I believe, in general, the fee is the predominating factor. Occasionally retention is aided, but usually only for a brief time.

(32) *It all depends*, and when *so depending* is good practice.

(33) I view the construction of large restorations, that call for the use of pulpless teeth as abutments, with shivers running up and down my spine.

(35) Providing abutments do not carry or cause dangerous foci, systemic conditions considered, pulpless teeth may carry extensive restorations. Of course, a very mild infection to an abutment would be admissible only with successful corrective local work and high systemic resistance.

(36) Bad practice.

(37) They may be constructed until pulpless teeth are *proven* to be a menace to health.

(38) Restorations to supply missing teeth should be limited to correct mechanical principles, which include extent; and the use of pulpless teeth, with a satisfactory periodontium in each instance, as abutments, is decidedly indicated. Assuming that the future of pulpless teeth is questionable, they can supply no greater service than as bridge abutments, thus also preventing mutilation of other teeth.

(39) It is not a question of "pulpless," "vital," or "non-vital" teeth. The question is: Are these teeth diseased and to what extent does this disease affect the health of the patient? I am sure there are large restorations on vital teeth used as abutments, and that these teeth are not diseased as a consequence.

(40) I do not believe in restorations that call for the use of pulpless teeth as abutments.

(41) *We don't make them.*

(42) I suppose this refers to fixed bridges, which I do not favor for large restorations under any circumstances.

(45) From personal experience, I feel that the use of pulpless teeth as abutments for large fixed restorations is not good practice. I should prefer not to use pulpless teeth as abutments for removable bridges, although I do not feel that the objection is as great as it is for fixed work.

LI. *What is your opinion on the use of large stationary bridges cemented to vital-tooth abutments?*

(1) I think when skilfully done, in a sanitary manner, it is the best thing for the patient. Preserve all teeth as long as possible, and let plates be the very last resort.

(2) It is not good practice. In the first place, they are unhygienic; secondly, they are apt to be mechanical failures in a short time.

(3) I am not in favor of large bridges cemented to any teeth.

(4) It has been my experience that, if a large stationary bridge is not a nuisance in one way, it is in another. They are filthy and unsanitary in most mouths. Pressure on one part causes mobility of the entire fixture; and in time this irritation causes disease of the supporting membranes, whether the abutments are vital or not. If the vital tooth has been sufficiently ground to make a well-fitting crown, the irritating cement, the shock of grinding, or whatever cause, induces an uncomfortable hyperemic condition, or complete devitalization; and for that reason I never employ them, except in cases of repair or remaking.

(8) They are of questionable safety, and each will probably have only a short period of usefulness.

(9) They have given good service in countless cases, and I consider it good practice.

(10) I am convinced that large fixed bridges eventually induce pathological conditions whether cemented to vital or to pulpless teeth.

(11) I regard as questionable practice the cementing of large stationary bridges either to teeth or to roots of teeth. (Oh dear! My questioner has half-backsliden by referring to "vital" teeth.)

(12) I do not approve their use.

(13) Unfavorable opinion.

(14) If the abutments are in good shape, I believe many of these bridges can be very successfully constructed, and will give excellent service.

(15) It is doubful whether they can long be thus employed and the roots to which they are attached remain healthy.

(17) Stationary bridges are unsanitary and should not be used if it is possible to employ another type of reconstruction. If used at all, the anterior portions of the arches should be selected, and the abutment teeth should be left vital.

(18) The use of large stationary bridges cemented to vital teeth is open to the objection of traumatism, which may result in the death of the pulps in some of the abutments. However, the practice is not so bad as it may seem. While the pulp is alive the patient is free from

that type of focal infection, and as a rule they do good service. Very heavy pressures of any fixed type are counter-indicated. I do not by any means consider that the practice of using vital teeth for abutments is a bad one.

(19) I do not think or believe that extensive stationary bridges are indicated.

(20) I disapprove of large stationary bridges cemented to vital-tooth abutments. I use a plate in preference to large bridges.

(21) They are not desirable, if they can possibly be avoided, and usually they can. If necessary, they are "not so bad," provided such bridges are given an abundance of support.

(22) All artificial appliances have their objections. Cleanliness is one great factor to keep in mind, but plates are rarely very clean, and they cover soft tissues. I think that, in certain cases, large bridges may be constructed so that they can be kept clean, even if stationary, though removable bridges would often be better.

(23) It is a question purely of oral hygiene. The patient needs instruction in exact cleansing of such bridges. Stationary bridges should be constructed with *convexed* cervices, etc., so that floss drawn along them will really cleanse, and not leave food debris, mucus, etc., attached. Special brushes will do much in this direction. Granting this, I see no objection to four- or five-tooth bridges, including abutments, on one side, provided the teeth are suited to them and not already loose; if they are, more looseness will result. If two sides are involved, two bridges, with or without an overarch bar, or removable bridges, on vital teeth, seem to me to be correct practice.

(25) It should not be attempted.

(26) As a general proposition it is not advisable. There might be an occasional exception.

(28) Their longevity is limited, but movable bridges and partial plates are very destructive to the teeth to which they are attached, and such appliances may be styled *temporary*.

(29) They are simply abominable. (No statistics.)

(30) I would advise use of a *removable* appliance.

(31) I am opposed to large stationary bridges under any conditions, as being unhygienic and in time unsatisfactory.

(32) "It all depends."

(33) I commence to feel uneasy when large stationary bridges cemented to vital-tooth abutments get as large as a six-tooth bridge.

(35) There are no objections to large fixed bridges on vital abutments, providing prophylactic construction and sound mechanics are insured, without endangering pulps by deep cutting.

(36) If based on sound engineering principles, vital-tooth abutments should not suffer.

(37) These constructions fail because the abutments loosen.

(38) The cementation to vital abutments, of bridges constructed upon correct principles, is the best method of supplying missing teeth in many cases. Fixation is not the important factor. Excessive stress from fixed bridges, removable bridges, clasp dentures, or traumatic occlusion, impairs the attachment of any tooth.

(39) Large fixed or stationary bridges are becoming rare in my practice. I presume they are not being used as they were at one time. I have seen instances where abutments remained free from periapical disease, under large bilateral bridges that had been worn for years. Removable restorations are much to be preferred. Here again, disease is a matter of non-resistance to one or more of the forms of injury: mechanical, chemical, and bacterial.

(41) *We do not do them.*

(42) "Bad" expresses my opinion.

(45) I am opposed to large stationary bridges on either vital or non-vital teeth. Not only is the average fixed bridge unsanitary, but it also places too great a strain on the abutment teeth, and prevents these teeth from having physiological movement.

LII. *In case you had a pulpless tooth and a vital tooth proximating, with the pulpless tooth apparently free from infection and with a space adjacent to the pulpless tooth due to loss of teeth: Would you remove the pulpless tooth and carry the bridge to the vital tooth, or not?*

(1) I prefer to anchor abutments on living teeth, when it is necessary to make a bridge anyhow.

(2) Not if the pulpless tooth were in good condition.

(3) Not if the radiograph showed no signs of apical involvement, and the roots could be filled to the apices.

(4) I cannot conceive an excuse for the removal of a perfectly good, non-infected, pulpless tooth, in order to replace it with an artificial substitute.

(8) Not if all other conditions were favorable.

(9) I would not.

(10) The patient's financial ability to pay for the treatment of the pulpless tooth, and to underwrite the chance of failure of the bridge through subsequent disease of the pulpless tooth, would be a factor to consider. Ordinarily I would use the pulpless tooth for an abutment, if I were convinced that my treatment was successful. In other words, I consider that the vital tooth is more apt to remain healthy than the pulpless tooth, but that the pulpless tooth may safely serve for many years.

(11) I should use the pulpless tooth, and adjust to it some form of removable bridgework.

(12) No, I'd be thankful for the chance to use the pulpless tooth and so avoid cutting the vital one.

(13) Not if I could make suitable use of the pulpless tooth, provided I could properly fill its root canals.

(14) If the pulpless tooth were in first-class condition and the patient in good health, I would use it; otherwise, I would extract.

(16) If the pulpless tooth were free from infection, I would retain it.

(17) The pulpless tooth should be retained and used, providing the periodontal tissues are in good condition, the radiogram is negative, and the tooth can be properly prepared as an abutment.

(18) If the patient were exhibiting no symptoms of focal infection, I would not remove the pulpless tooth but use it as an abutment, providing I felt confident, from the radiographic standpoint, the history, and all available means, that it was not liable to become a focal-infection tooth. Of course, if the patient were one whose family history showed a tendency toward cardio-renal disease or any of the chronic diseases resulting from focal infection, which might indicate that there was a lack of family immunity or resistance to the organisms causing that type of disease, I would remove the tooth as a matter of prevention.

(19) No.

(20) Several factors would enter into the decision: the health and age of the patient; the strength of the approximating vital teeth; the strength of the non-vital tooth; and the size of the space. If the patient were in good health, I would retain the non-vital tooth (if

the space were to be bridged), providing it was strong enough to act as an abutment. If the patient displayed symptoms suggestive of focal infection, I would remove the non-vital tooth. With a normal healthy patient I would be very anxious to save the non-vital tooth, and to use it as a bridge abutment, rather than partially to strip the enamel from a vital tooth for a bridge abutment. If the space was one requiring a plate, I would extract the non-vital tooth.

(21) If the canals of the pulpless tooth are properly filled and there is no history of disease, I would attach a bridge to a pulpless tooth and avoid mutilation of a vital one.

(22) I should use the pulpless tooth.

(23) On general principles, I would extract the pulpless tooth and carry the bridge to the vital tooth.

(25) No, I would not. No bridgework is permanent. If we could have three to five years' use with the pulpless tooth, we would add just that much to the utility of bridgework for that patient.

(26) I would not.

(28) I would not.

(29) If *I* had the case, as directly questioned, I certainly would not. (No statistics.)

(30) I would not advise such a procedure.

(31) If the pulpless tooth gave enough clinical and radiographic evidence of being healthy, I would use it by all means.

(32) I would not.

(33) If it did not make the bridge too big, nor the span too wide, I would extract the pulpless tooth and attach the bridge to the vital tooth.

(35) With no apical infection, and with periodontic conditions sound, or capable of being so made, the pulpless tooth could safely be used as an abutment, and there would be no reason either to remove it or to cut into the approximating vital tooth.

(36) With a good history, and the possibility of careful sterilization and refilling of the canals, I would not hesitate to use this tooth for a pin-inlay attachment, or a porcelain-crown attachment, or as one end of a fixed or removable pin-inlay bridge with the other pin-inlay attachment in the vital tooth, if the strategic value of such a root, or the esthetic importance of retention of that root, had great significance

in the case of that particular patient. The fixation of such a root in the anterior region of the mouth, with the maintenance of labio-alveolar contour, is very important where there is absence of systemic infection and where no banded crown is used.

(37) No.

(38) No. Assuming the future of a pulpless tooth to be more doubtful than that of a vital tooth, pulpless teeth, apparently free from infection, should be utilized rather than to jeopardize the safety of other teeth.

(39) The history of the pulpless tooth would be the determining factor. If, at any time, it had an attack of pulp infection with suppuration (even if there were no evidence, clinical or radiographic, of present disease), discretion and respect for potentialities would make me inclined to sacrifice the tooth to the forceps rather than run the risk of having a recurrence of infection in tissues no longer quite normal. If the tooth had never been septic, having had, for instance, a pulpitis or an accidental pulp exposure as the reason for pulp extirpation; or if it had been filled under aseptic precautions—such a tooth, being free from disease, is as good a tooth as another to hold a correctly-made abutment crown for a bridge.

(41) I certainly would not remove the pulpless tooth but would use it, if I adopted that method of restoration. If the operation were successful (say, for ten, fifteen, or twenty years) and the tooth then gave way, the tooth could then be removed and the vital tooth used. In the meantime, the patient might have died and gone to "heaven," where it would be desirable to go with as much of the original body as possible.

(42) If only one tooth were missing (or not more than two), I would not hesitate to make use of a pulpless tooth, free from infection, as an abutment. Beyond that number (2) of teeth to be replaced, I prefer a plate or removable bridgework.

(45) If the patient were in good health, and willing to have me perform an aseptic root-canal operation on the non-vital tooth, I should use the tooth as an abutment. If the patient were not in good health, I should extract the pulpless tooth and carry the bridge to the vital tooth.

III. QUESTIONS RAISED BY CORRESPONDENTS IN RESPONSE TO THE GENERAL INVITATION, TO THEM, TO "SUGGEST ANY OTHER QUESTIONS YOU THINK SHOULD BE ANSWERED."

LIII. *Please enter here any question you would like to have answered.*

(1) Why is it so hard to get the profession generally to take any interest in preventive dentistry, oral prophylaxis, and the treatment of Riggs' disease? This is one of the greatest services that the dental profession can render to humanity. The world wants it, needs it, and is willing to pay good fees for it.

(2) "What's the use?"

(3) (a) I should like to know, definitely, whether Buckley's desensitizing paste really kills pulps, and the arguments in support of claims for or against it.

(b) How have advocates of extraction of all pulpless teeth arrived at their conclusions?

(4) A woman sixty-seven years of age has had one severe, and subsequently two or more milder, apoplectic strokes. Her tongue and right hand are paralyzed. She wears a full upper plate, and a partial lower plate; eats well, sleeps well, and otherwise is physically perfect. The six lower anterior teeth (one more or less loose and diseased) are a hindrance in eating and unsanitary. The family wish the teeth removed. Would you extract them? Would you use conduction or infiltration anesthesia for the cuspids?

(8) What will be the next dental "rainbow"?

(10) (a) Can apical cementum remain vital in periapical disease?

(b) Can apical cementum once infected be sterilized, and, if so, by what agency?

(c) In case check-up radiographs evidence complete regeneration of alveolar bone about a root apex, would you expect the lamina dura to be reformed?

(d) Is the absence of the lamina dura in such cases (c) evidence of persisting infection?

(e) What technic of pulp extirpation prevents instrumental injury to the apical pericementum?

(f) What sometimes causes persistent lameness in a tooth from which a vital pulp has been removed, where the filling does not project

through the apex, and there is never any radiographic evidence of periapical abnormality?

(g) What method of taking bacterial cultures prior to canal filling do you consider most reliable and scientific? Why?

(11) When will the profession "forget" the terms "non-vital" tooth, "dead" tooth, and "devital" tooth (there being "no such word" as devital), and realize that the extirpation of a pulp does not destroy the vitality of the most essential part of a tooth, its cementum? Teeth possessed of pulps carry non-vital enamel. Pulpless teeth carry non-vital enamel and non-vital dentine. The same condition is obtained with the layer of cells in the integument over the entire body, this also being non-vital. If we must be shed of all non-vital structure, such as enamel and dentine, we should then also have our bodies scraped until they bleed, so as to reach vitality. A callosity is dead scar tissue, and we who work with our hands still live.

(13) (a) What justification has anyone for taking the stand that all pulpless teeth should be removed?

(b) What justification has anyone for basing a dental diagnosis solely upon what he thinks he sees in an x-ray picture?

(c) What justification has a physician for issuing instructions to a patient or to a dentist regarding details of dental treatment for said patient, without having had consultation with a competent and experienced dentist?

(23) Why does not the profession stay at home and give up its national and state annual conventions for one year only; devote its traveling, hotel, and frivolous expenses, in these relations, plus three days of earnings in the office, *to the Research Fund*, which could be increased to $2,000,000 by such action; and then expend this sum on a corps of *experts* to conduct physical and chemical researches to determine what we can or cannot do; then inform the public; and thus enable us to proceed on an *honest basis?* (My own answer to this question is not fit for publication.)

(25) (a) How may I fill perfectly a root canal so that the tooth may have the greatest protection?

(b) Is pressure anesthesia harmful?

(29) (a) Is it not a fact that a tooth has two sources of vitality—one, the pulp; the other, the periodontal membrane?

(b) Is it not also a fact that *pulpless* teeth—called, throughout the questionnaire, "non-vital" teeth—are really vital, and that the term *non-vital* as applied to such teeth as are here considered, is scientifically incorrect and should not have been used? ("No statistics.")

(31) (a) What should be the primary function of dentistry with regard to pulpless teeth?

(b) Does the literature of the past century give evidence of such a wide-spread controversy, during any previous epoch, as now prevails with regard to the subject of treatment of pulpless teeth?

(32) I surely wish I had time to do so.

(33) (a) Is there any one in the dental profession who will not agree that the dentistry of the future must be a dentistry devoted to the prevention of toothache, i.e., the prevention of dental disease from ever passing beyond the truly dental stage—the stage of dental caries? When dental disease passes into the pulp, it becomes a dento-vascular disease. When it goes beyond the end of the root, it becomes an osseous disease, and it may then become a disease of any part of the body.

(b) I believe it is true that all dentists see the necessity for finding and filling carious cavities before they reach the pulp. If this is true, why don't we as a profession try specifically to teach the public effectively about this necessity?

(c) Why don't we as a profession develop a technic of mouth examination by the employment of which we could really find all cavities?

(37) Why is it that conditions enumerated below are commonly called by *all classes* of dentists by one name:

(a) Loose teeth, gums receded, no tartar.

(b) Loose teeth, no recession, no deposit.

(c) Firm teeth, recession, deposit.

(d) Firm teeth, no recession, deposit.

(An endless chain might be added by mentioning each of the above in combination with pain present, discoloration of gum tissue, spongy gums, *and the converse.*)

(45) (a) In the present state of our knowledge of the histology of dentine, and the histology and physiology of cementum, is it possible to remove the pulp of a tooth and fill the root canals so that the peri-

apical tissues will not become infected within a comparatively short time?

(b) Until our knowledge has developed so we can safely treat pulpless teeth in a manner that will prevent subsequent infection, are we justified in taking a chance with the public health by treating non-vital teeth?

(*To be continued*)

933 Metropolitan Bank Building

A MICROSCOPIC STUDY OF CANCELLOUS BONE AT THE ROOT ENDS OF DEAD TEETH

THE RESULTS OF WORK DONE IN COLLABORATION WITH FRANK E. BLAISDELL, M.D., ASSOCIATE PROFESSOR OF SURGERY, DIRECTOR OF THE LABORATORY OF SURGICAL PATHOLOGY, LELAND STANFORD, JR., UNIVERSITY MEDICAL COLLEGE, SAN FRANCISCO, CALIFORNIA

JOSEF NOVITZKY

San Francisco, California

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I. INTRODUCTION

The investigations that resulted in this paper were first undertaken in order to determine whether drainage from the root end of a dead tooth should be regarded as drainage directly into the blood stream. It was our purpose to examine the venous system of marrow, and also of the spleen, with the idea of discovering whether the blood was confined wholly in vessels, in certain parts of red bone-marrow and of the spleen, or whether it was free in the interstitial spaces. It was our purpose, in particular, to determine whether there was free blood in the reticulum of the red marrow of the jaws or whether the smaller veins had such delicate walls of endothelium as to render those walls practically non-existent.

This purpose was abandoned, however, for a more important one, when the first slides to be examined showed the presence of fibrous marrow in the cancellous bone at the root ends of dead teeth. Previous investigations had shown infection in cancellous bone at the

root ends of dead teeth in all cases examined; but the evidence of infection had been disregarded by many dentists, who still maintain that devitalized teeth can be retained uninfected in the jaws. It now became our purpose to determine the validity of the earlier evidence of infection, in terms of the new evidence offered by the presence of fibrous marrow in cancellous bone at the root ends of dead teeth.

The presence of fibrous marrow in all cases would indicate infection in all cases. It cannot be argued that fibrous marrow would indicate mere irritation and not infection. Investigations have shown that permanent fibrous marrow does not result from mere irritation. A nail driven into cancellous bone under aseptic conditions at first causes the marrow in the region of the puncture to become fibrous. If, however, asepsis is maintained, the results of the irritation are resorbed and the marrow becomes normal.

II. GENERAL OUTCOME OF THE RESEARCH

Our investigations showed clearly the presence of fibrous marrow in cancellous bone at the root ends of dead teeth in all cases examined. This gives us further (in fact, *final*) evidence of infection in or from devitalized teeth. The normal marrow was conclusively demonstrated to be both lymphoid and fatty.

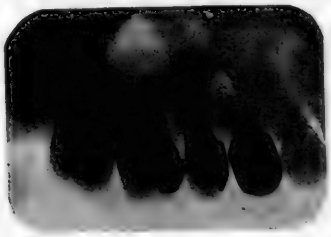
It was noted, in all cases examined, that fibrous marrow was most abundant in the *apical* regions of dead teeth and in many instances restricted to such regions, indicating that the septic irritation was apical rather than gingival. The infection, in other words, gains access to the cancellous bone from the dead-tooth dentine by way of the apical foramen and not, as some have thought, from the mouth through the gum flap into the tooth socket.

In the cases of all the dead teeth examined, a diagnosis of myelitis was established microscopically by the presence of fibrous marrow, a deviation from the normal, in the adjacent cancellous bone of the jaws. The presence of fibrous marrow indicated not fleeting but permanent irritation, due undoubtedly to reinfection from the dead tooth. This fact is often overlooked—the menace of dead teeth lies not so much in infection as in constant reinfection.

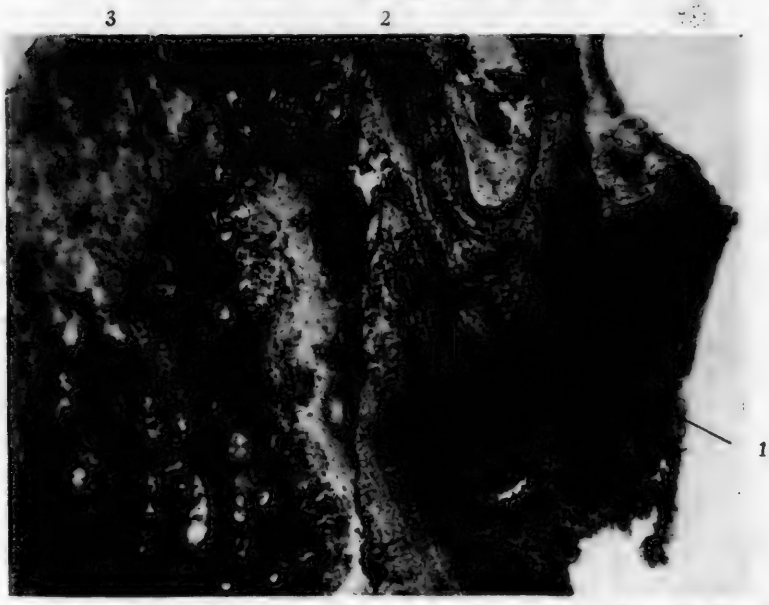
III. HISTOLOGIC AND DIAGNOSTIC DATA ON WHICH THE CONCLUSIONS ARE BASED

The accompanying photographs and microscopic diagnoses, made by Dr. Blaisdell, illustrate the facts.

Case 556



CASE 556: L. superior. Roentgenogram. See slide 556



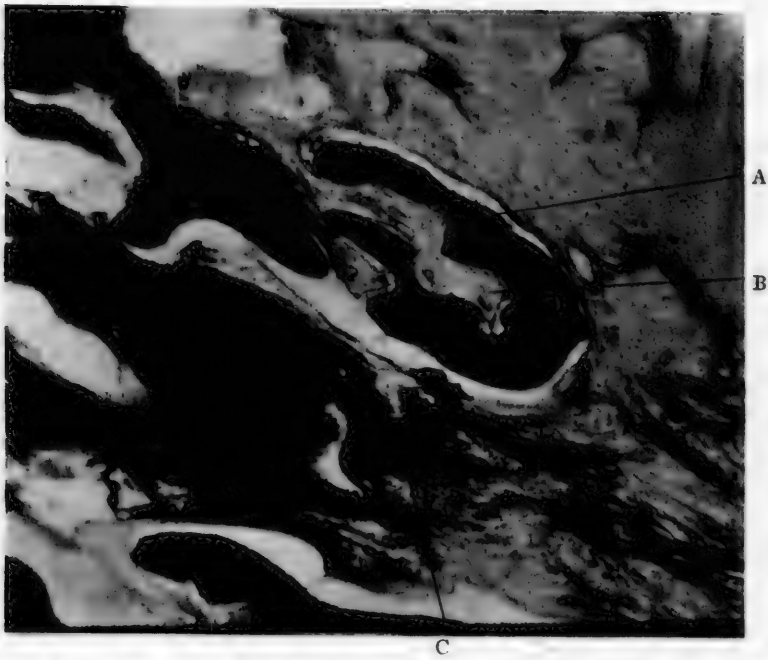
SLIDE 556: L. superior. *1*, necrotic bone. *2*, hemorrhagic area. *3*, round-cell and leukocytic infiltration. [Obj. 2 in.; oc. 4; bellows length, 14 in.]

The above paraffin section (Case 556) showed inflammatory tissue and necrotic bone. There were some edema, granulation tissue,

increase in fibrous tissue in places, and in some parts hemorrhagic exudate. There were few plasma cells, and many leukocytes were intermixed with round cells.

Case 461

CASE 461: antral wall. Roentgenogram. See slide 461



SLIDE 461: antral wall. Shows well-stained osseous trabeculae and fibrous marrow. *A*, deeply stained trabeculae; lacunae with bone corpuscles abundant but not shown. *B*, fibrous marrow. *C*, denser fibrous marrow. [Obj. 1 in.; oc. 1 in.; bellows length, 12.5 in.]

The specimen for examination (Case 461, antral wall) consisted of prepared histological slides showing paraffin sections mounted in balsam, and stained with hematoxylin alone, hematoxylin and eosin, and hematoxylin and picro-fuchsin. Microscopical examination showed well stained osseous tissue and much fibrous marrow. The bone trabeculae contain numerous lacunae, which are occupied by well stained bone cells; in several small areas these cells are absent and the lacunae are empty; the lamellization is partly distinct. Some of the marrow spaces are filled with fatty marrow. The fibrous marrow varies in density; in one area through the section it is dense and has stained deeply. Capillaries are very abundant in the fibrous, but not in the fatty, marrow. There is a very diffuse and sparse round-cell infiltration in the fibrous, and in some parts of the fatty, marrow; scattered plasma cells and endothelial leukocytes are seen. About several of the larger blood vessels are denser accumulations of round cells. *Diagnosis:* Chronic inflammatory tissue and myelitis.

Case 482

CASE 482: old abscess or "cyst." Roentgenogram. See slides 482

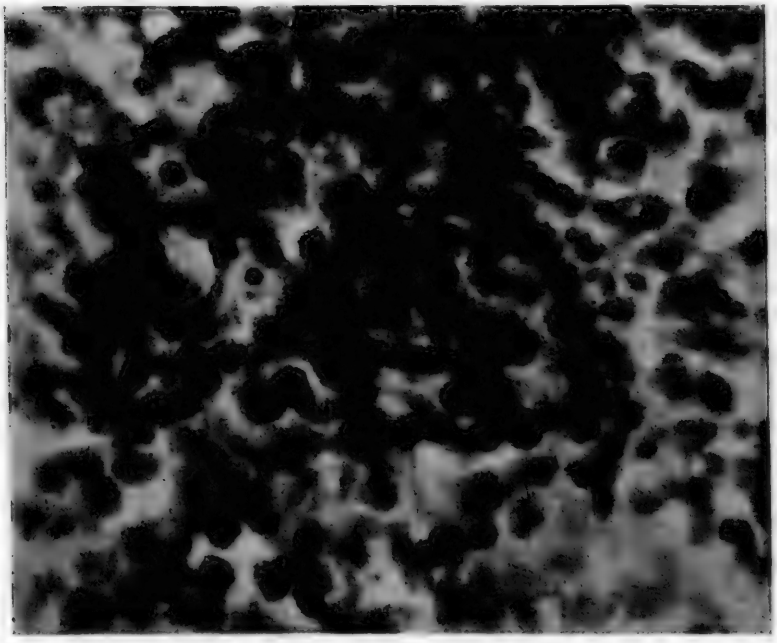
The roentgenogram of the left side of the mandible (Case 482) indicates a large region of less than normal density surrounded

by eburnated or sclerosed bone at the site of the first-molar. The tooth, described by the patient as "ulcerated," had been extracted six years previously. The two photomicrographs show what appeared at microscopical examination of the tissue (indicated by the arrow) when it was removed by an operation.

SLIDE 482, I: old abscess. *A*, newly formed connective tissue. *C*, granulation tissue. *D*, purulent center. *E*, hemorrhagic exudate. See slide 482, II.

The paraffin sections of Case 482, diagnosed as chronic inflammatory tissue and granulation tissue with abscess, show a few osseous trabeculae differing in size. These have stained well. The lacunae contain deeply stained bone cells. There is a large amount of newly formed connective tissue, infiltrated with round and plasma cells. Around this area the collagen bundles are abundant; in the inner part, the tissue is quite cellular. Leukocytes, intermixed with round and plasma cells, increase in number from the periphery to the center.

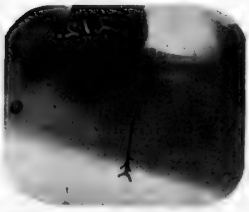
A cavity filled in with partially calcified granulation tissue (as shown in the roentgenogram of Case 482) is often confused by dentists with physiologic ossification. But it is only necessary to see a few cases at operation to realize that there is no similarity between the deposit of calcic debris and a physiologic ossification.



SLIDE 482, II: old abscess. Purulent center, more highly magnified. See slide 482, I. [Obj. A (Zeiss); oc. 4; bellows length, 16 in.]

Case 482 proved to be an abscess. The term "cyst" is used frequently to designate a suppurative area in the vicinity of the root end of a dead tooth. Common findings indicate that *abscess* would be a more accurate term, as the "cyst" wall is generally composed of granulation and fibrous tissue. The term "cyst" is more accurate when epithelium surrounds the mass; but, even in this case, suppuration may be superimposed on the cystic condition.

Case 471



CASE 471: fragment of bone from a so-called "cyst." Roentgenogram. See slide 471

SLIDE 471: fragment of bone from a so-called "cyst." Shows a fragment of bone taken from curettings. The tissue has not stained well nor evenly, and the lacunae are empty. The accompanying material showed granulation tissue and intermixed leukocytes. [Obj. A (Zeiss); oc. 4; bellows length, 13 in.]

Case 471 shows a roentgenogram and a photomicrograph of a fragment of bone taken from a case of so-called "cyst." There is shown a large area representing what appeared to be structureless calcic debris in the molar region on the left side of the mandible. An

abscessed tooth had been extracted from this region some years before operation. At operation the curette slipped into a hole in the cancellous bone. Granulations and odor were noted. The microscopical examination, by Dr. Blaisdell, of tissues mounted and sectioned in paraffin, showed broken-up cancellous tissue and fibrous marrow, intermixed in some places with leukocytes. The osseous trabeculae have stained rather poorly. Lacunae and the haversian canals are uniformly empty. The case was diagnosed as infective osteomyelitis.

In connection with these cases it is interesting to call attention to the so-called "bone whorls" that are mentioned in dental writings. One of these is pictured as Case 37.

Case 37

CASE 37: bone whorl. Roentgenogram (no accompanying "slide")

This case would probably prove to be eburnated bone at operation. In cancellous bone, eburnation due to embolism, eburnation due to local sepsis, and partially calcified granulations, appear, at times, to be similar in roentgenograms and should be differentiated clinically. The first of these three is undoubtedly due to circulatory arrest. The atrophy of marginal alveolar process indicates, as did also the clinical data, that systemic causes were responsible for the blocked blood vessels.

A "bone whorl" may be regarded as an embolic circulatory arrest resulting in a deposition of blood salts in the reticulum of cancellous bone and shown in the roentgenogram as an eburnated area. In Case 37 the atrophy of marginal alveolar process indicates, as did also the clinical data, that systemic causes were responsible for the blocked blood vessels.

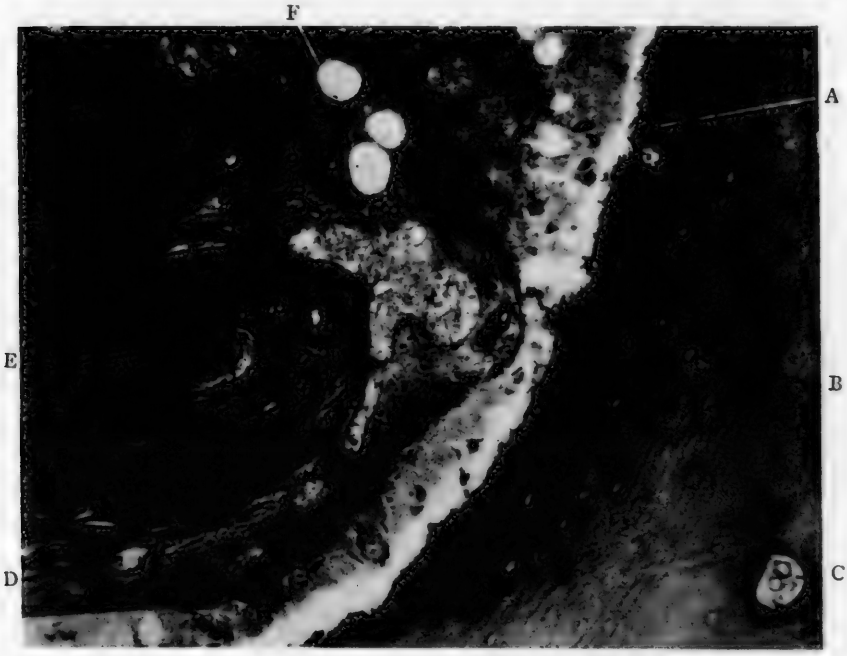
An eburnated region in cancellous bone and a region of partially calcified granulations are difficult of differentiation in a roentgenogram. The term "bone whorl" for such a formation is quite lacking in diagnostic value. If an embolus has caused a circulatory arrest in cancellous bone, and a resultant obliteration of haversian spaces by blood salts has followed, other evidence of atrophy or sclerotic bone is usually found. If a partially calcified granuloma is present, the clinical history and the roentgen-ray evidence are usually sufficient to differentiate it without recourse to a meaningless term like "bone whorl." The granuloma, at operation, will be found to occupy an offensively odorous cavity in the bone. Case histories indicate that a granuloma has persisted for thirty years after the extraction of an abscessed tooth, with disastrous results to the health of the patient.

Case 551

CASE 551: old region remaining after extraction of a tooth. Roentgenogram. See slide 551.

In the examinations of cancellous bone at the root ends of dead teeth, a diagnosis of osteomyelitis was commonly possible. This was established by the absence of bone cells in the lacunae, by irregular lamellae, by a differentiation in the staining quality, and in the more extreme destructions by the presence of structureless necrotic debris. The roentgenograms and photomicrographs of Cases 551 and 555 show cases diagnosed as osteomyelitis.

In Case 551 is shown an old mass remaining after extraction. At operation a rounded depression was seen in the outer plate of alveolar process. This was removed with a gouge. A microscopic examination of parlodion sections of it showed cancellous bone. The marrow spaces were filled with fibrous marrow; in some places a few fat cells were seen but no lymphoid marrow. Cellular infiltration was sparse,



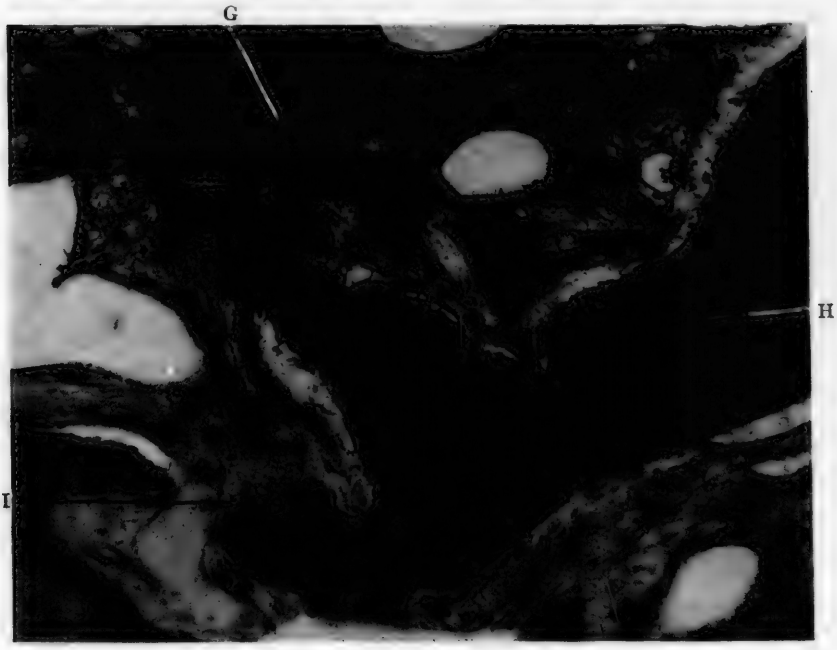
SLIDE 551: old region remaining after extraction of a tooth. *A*, newly formed bone with cells in lacunae. *B*, dead bone; lacunae empty. *C*, haversian canal; tissue shows retrograde changes. *D*, blood-vessel. *E*, dense fibrous marrow. *F*, empty fat cells. [Obj. A (Zeiss); oc. 4; bellows length, 13 in.]

and consisted mainly of plasma cells and fewer round cells. There were many blood vessels, ranging from those of moderate size to smaller arterioles. The osseous trabeculae showed some variation in staining. The lacunae were empty in numerous areas, but in others bone cells were persistently present. In certain trabeculae the tissue containing empty lacunae has stained more heavily with hematoxylin.

This tissue was surrounded by newly formed bone in which bone cells filled the lacunae. The darker areas appeared to have been buried by a productive or condensing osteitis, for their margins were abrupt and irregular against the even haversian systems of the new bone. In places, osteoblasts lined the margins of the trabeculae; about other parts of the bone, osteoclasts were observed.

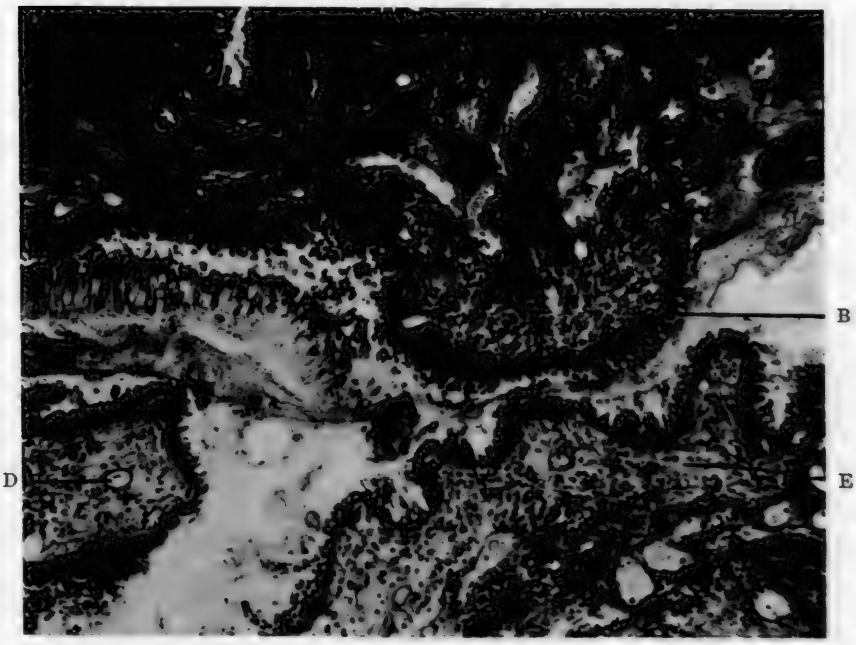
Case 555

CASE 555: necrotic antrum-floor (teeth extracted some years previously). Roentgenogram. See slides 555, I, and 555, II.



SLIDE 555, I: necrotic antrum-floor. *G*, shows abundant fibrous marrow, with a few scattered plasma cells. *H*, trabecula of live bone, with cells in lacunae and margined with osteoblasts. *I*, three osteoclasts. [Obj. A (Zeiss); oc. 4; bellows length, 13 in.]

Under Case 555 are shown a roentgenogram and photomicrographs of a section of antral membrane and a section of cancellous bone. This case was diagnosed as chronic osteomyelitis with inflammatory thickening of the antral membrane. The roentgenogram shows the necrotic antral floor some years after the tooth had been extracted.



SLIDE 555, II: necrotic antrum-floor. View of antral membrane. Plasma-cell infiltration marked; also increase in the fibrous tissue, especially in the corium of the mucosa. *B*, epithelial surface. *E*, edematous connective tissue. *D*, blood vessels. [Obj. A (Zeiss); oc. 4; bellows length, 13 in.]

Slide I shows extensive round- and plasma-cell infiltration, with increase in the fibrous tissue, especially in the corium of the mucosa, with edema of the tissue lining the floor of the maxillary sinus. Slide II shows an abundance of fibrous marrow with a few scattered plasma cells.

We should call attention here to a point brought out in 1915,[1] namely, that a diagnosis of osteomyelitis, in severe chronic cases involving the nutrient tubes and canals of the jaws, is not very helpful, since it ignores serious complications that could not be alleviated by an indefinite surgical treatment, such as that in surgical interference in osteomyelitis. For instance, suppuration and death of the contents of the inferior dental canal should be specifically diagnosed rather than cloaked with the term osteomyelitis, which does not indicate the exact region involved.

The cases studied in our examinations of cancellous bone would serve well to point out to the surgeon, unfamiliar with localized ills resulting from dead teeth, that infective elements remaining after the extraction of septic teeth cannot safely be ignored. There is no sufficient reason for believing that such infective elements are not harmful to the body, even though the danger of reinfection that existed when the tooth was in the jaw has been lessened by removal of the tooth The residual infective elements are not exfoliated. They remain as pathologic regions that become surrounded by bone more or less sclerosed and eburnated. This is shown very clearly by the facts pertaining to Case 482.

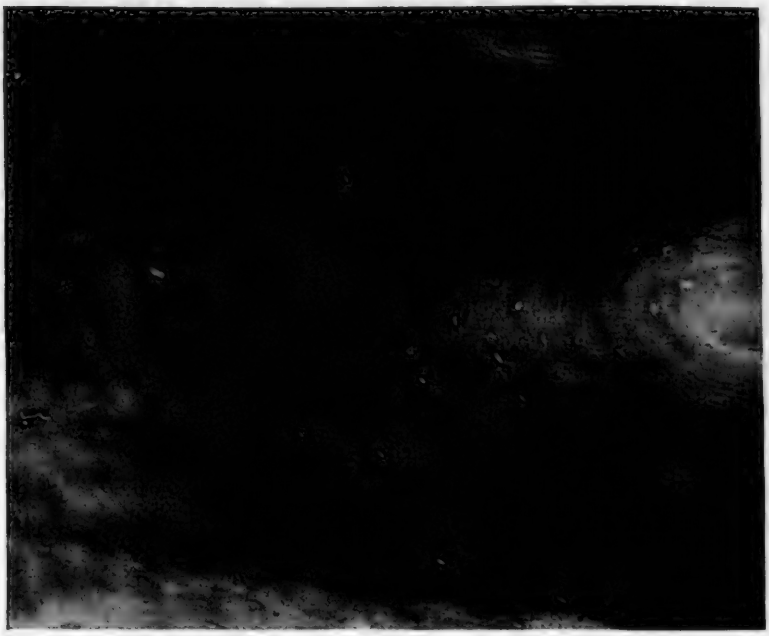
Case 401

There has been no thoroughly satisfactory explanation of the lack of sequestra in the cancellous bone at the root ends of dead teeth, although it has been suggested that this lack is due to the very abundant blood supply in and about the jaws. Less vascular regions, such as the long bones, where the blood vessels are not so numerous, and where they would be more apt to become occluded from inflammatory swellings, are regions where sequestra are commonly found.

Although sequestra in the cancellous bone at the root ends of dead teeth are practically unknown, a minute sequestrum, as an exception, has been occasionally observed. Case 401 was a very interesting one. A first molar was removed by operation, but the operative field did not extend to the lingual plate, as normal cancellous bone was seen to be lining the inner side of the lingual plate. Ten months after this

[1] Novitzky: The radiograph in dentistry, *Pacific Dental Gazette*, 1915, xxiii, p. 265.

CASE 401: a small sequestrum on a lingual plate. Roentgenogram. See slide 401



SLIDE 401: a small sequestrum on a lingual plate. Shows osseous tissue that has stained irregularly and the lacunae are empty. Portions of two haversian systems are shown; these also show no bone cells. In another part of the tissue, however, bone cells are present in the lacunae. [Obj. A (Zeiss); oc. 4; bellows length, 13 in.]

first operation a small red spot was observed on the lingual side of the lower first-molar socket. Clinically there had been no acute inflammation outside of the small spot on the gum (see roentgenogram). After some weeks of observation, the mucous membrane, when raised and retracted, showed a wart-like protuberance of what appeared to be bone tightly adherent to the lingual cortical plate. The protuberance was removed with a chisel. This tissue was run through alcohol, mounted in parlodion, and stained. A microscopical examination of a 2 mm. section by Dr. Blaisdell showed osseous tissue that had stained fairly well but with irregular intensity. For the greater part, the lacunae were empty, with no signs of bone cells. About some of the haversian systems, however, between the distinctly concentric lamellae, bone cells occupied the lacunae. At the periphery of these haversian systems, the lamellae between which the lacunae were empty ended abruptly and irregularly. The slide shows portions of two haversian systems with lack of bone cells.

If the mass described above had been situated between the cortical plates, it would probably have broken down and become filled with granulation tissue. Partial calcification, permanent or temporary, might have followed. Such a mass sometimes does calcify. When observed in roentgenograms over a period of some years, changes in density are noted; apparently broken-down bone and blood salts are partially absorbed. New blood salts may be again thrown down, when there is a low-grade inflammatory change.

Many regions have been opened in edentulous jaws, years after dead teeth have been removed. In all of these from which cultures were incubated infective pathogenic microörganisms have been found. Odor and granulation tissue were noted. Microscopic examination showed round-cell and leukocytic infiltration of granulation and fibrous tissue. Edema, and hemorrhagic and purulent exudate, were noted. Examinations of osseous tissue showed fibrous marrow, empty bone-lacunae, and irregular lamellae bearing evidence of osteoclastic activity.

The fact that eburnated cancellous bone is so commonly found in the jaws indicates that an infectious process has been present. It has already been pointed out that if infection is not present, fibrous marrow is absorbed and replaced by lymphoid marrow. If infection is

not present, eburnation does not take place and the normal reticulum of cancellous bone is maintained.

Clinically there is much evidence of extensive eburnation following suppurations in cancellous bone elsewhere than in the jaws. But eburnation was found so commonly, in osteological sections of maxillary and mandibular bones, that, at first, it was thought that errors in technic were to blame. Bones to be sectioned were first thoroughly dried and infiltrated with melted paraffin. After twelve hours the chilled paraffin was cut away from the outside and the bone was cut into sections with a fine saw. A few days of immersion in zylol removed the paraffin from the cancellous bone as well as the surface of the bone. The specimen was then dried and examined. Eburnated areas were seen so commonly that it was exceptional to find a specimen that could be considered normal. These findings left no doubt of the fact that dead teeth are gateways for the entrance of infection into the interior of bone.

We have seen, clinically, regions of eburnation (indicated in roentgenograms) that were undoubtedly due directly to blood-borne disturbances. The number, however, showing clearly (from the region involved and from the position of contact with dead teeth) that infection had gained access through dead teeth, was extremely high in comparison with the number thought to be due to blood-borne infections.

IV. SUMMARY OF CONCLUSIONS

In conclusion, we may summarize our results in the statements that, in all cases examined, cancellous bone at the root ends of dead teeth was found to be infected; that it was characterized by the presence of fibrous marrow; and that it could be diagnosed as affected by myelitis or osteomyelitis. *This should serve as final proof of the long-disputed point that tooth dentine dies when the tooth pulp dies.*

The deposition of cementum on a dead tooth does not imply that vitality exists in the tooth any more than the fibrous encapsulation of a silk suture in the bodily tissues implies that vitality exists in the silk. The author replanted an extracted tooth *after boiling it*—it was then certainly *dead;* but two years later, when it was removed by

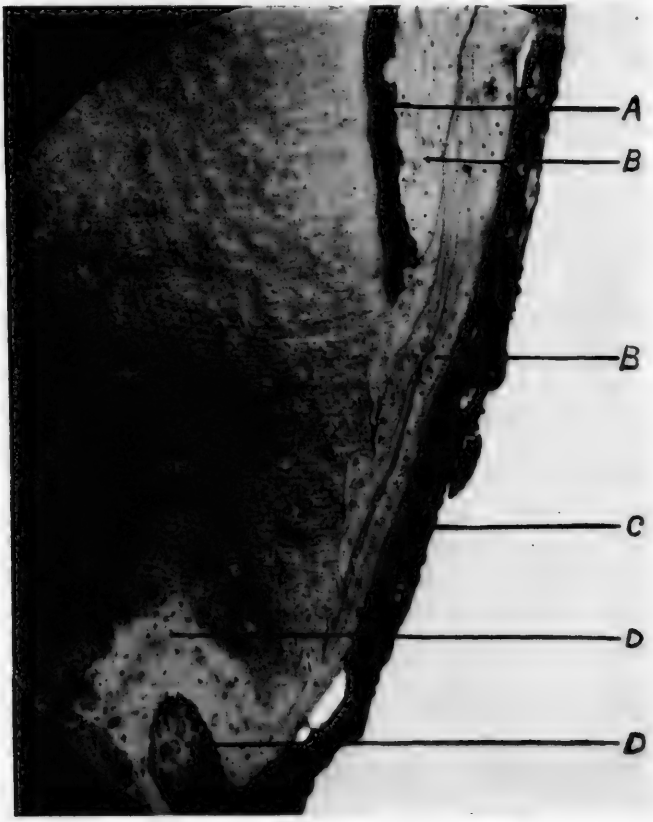
operation, sections of it under a microscope showed young lacunated cementum deposited on the old *boiled* cementum.[2]

The condition of the cancellous bone about the root ends of dead teeth offers final and convincing evidence of the fact that dead teeth are dangerous sources of infection, and that in many cases the danger cannot be removed by the old method of extraction.

Case of a replanted (boiled) tooth, I: section through the root. Granulations are partially detached from the root stump. An area of suppuration is seen at A. This tooth had been boiled for fifteen minutes before replantation. At operation two years later, the tooth was found to be thoroughly septic. (See *fig.* II of this case, herewith.)

[2] Novitzky: The problem of devitalized (dead) teeth, *Journal of Dental Research*, 1920, ii, p. 575.

The term "dead tooth," as used in this paper, signifies every tooth that is really lifeless, namely, *every tooth that is pulpless*. A pulpless tooth is dead, no matter what embalming process has been used on it. Living pericemental tissues cannot give vitality to dead teeth[2] any more than living green grass can give vitality to dead sheep. The statement that pulpless teeth are not dead, until the tissues around



Case of a replanted (boiled) tooth, II: section of the tooth illustrated in *fig.* I of this case, herewith. *A*, old cementum, deposited *before* replantation. *B*, lacunated cementum; deposited *after* replantation; here the layers of cementum are quite thick. *C*, pericemental structure; fibroblasts are seen clustered on the edge of calcic deposits—some are apparently submerged in the calcified tissue. *D*, a region of resorption, filled in by young cementum.

the root are also dead, might be carried to the logical conclusion that pulpless teeth are not dead until the entire body containing them is dead.

V. ADDENDUM: COMMENT ON A PAPER BY MERRITT

Since the foregoing was presented to this JOURNAL,[3] Dr. A. H. Merritt has published roentgenographs showing that changes take place about the root ends of infected teeth as a result of treatment that included sterilization and root filling.[4]

That such changes take place, there is no doubt. Dr. Merritt states that, although the exact nature of these changes is unknown, they indicate that an infected pulpless tooth can be *apparently* restored to health. He presents roentgenographs to disprove the statement that every devitalized tooth, six months after devitalization, is infected, no matter how it has been treated. We have here two distinct things: (a) evidence of change shown by roentgenographs; (b) opinion, not evidence, that such change means absence of infection.

It is necessary again to emphasize the fact that infection, fibrous marrow, sclerosed bone, or even dead bone-cells, cannot always be seen in a roentgenograph. The roentgenograph does not show infection; it generally shows sclerosed bone; while it may or may not show dead bone-cells. In some cases it does not show the difference between fibrous granulations and normal bone.

In order to demonstrate that an infected pulpless tooth has been restored to health, it is necessary to show (a) that cultures of the cancellous bone around the root end are sterile when incubated; and (b) that microscopic examination of the cancellous bone indicates normal bone and marrow, and an absence of edema and round-cell infiltrations. These things are not shown by the roentgenographic studies of the eight cases presented by Dr. Merritt.

To one familiar with the so-called "cures" of dead teeth, as seen at operation, any of the eight cases presented by Dr. Merritt would suggest the presence of pathological conditions. A glance at the

[3] Dr. Novitzky's paper was received shortly before the number containing Dr. Merritt's paper had been issued.—(W. J. G.)

[4] Merritt: Reparative changes that take place about the root ends of infected pulpless teeth, after treatment, *Journal of Dental Research*, 1921, iii, p. 229.

roentgenographs makes it safe to say that, in each of the cases purporting to indicate apparent restoration to health, a microscopic examination would indicate the presence of fibrous marrow, round-cell infiltration, and dead bone.

Publication of the evidence in support of the claim of Box, that a tooth has more than one source of nourishment, has been eagerly awaited. Long ago the writer believed that he himself had proved that a normal tooth in man has only one source of nourishment, and his belief has not changed. The writer showed, however, that, even if minute channels entered the tooth but not by way of the apical foramen, such channels would be insufficient for the maintenance of vitality in the tooth after the pulp is removed.

209 Post Street

COMMENT ON NOVITZKY'S "MICROSCOPIC STUDY OF CANCELLOUS BONE AT THE ROOT ENDS OF 'DEAD TEETH'"[1]

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I have been given an opportunity to read the manuscript of the foregoing paper by Dr. Novitzky and have been invited to prepare a statement regarding it for simultaneous publication.

I consider Dr. Novitzky's paper an excellent study of regions of eburnated bone in the jaw, and also agree with his interpretation of the microscopic findings. I should say, however, that the paper does not come up to the implications of the title: "a microscopic study of cancellous bone at the root ends of dead teeth." There is only one instance where the bone surrounding the apex of a pulpless tooth has been studied, namely, that in fig. 556. All the other specimens are taken from cases where teeth had been previously extracted and a region of eburnated bone located by roentgen examination. In this one case, fig. 556, the first bicuspid in question shows roentgen evidence of the presence of a large region of decreased density in the bone. Such conditions have been studied abundantly, and no one doubts that tissue taken from such a region shows an infection.

The conclusions drawn in the paper, however, would lead the reader to believe that Dr. Novitzky condemns all pulpless teeth as sources of infection, *no matter whether the roentgen examination shows improvement due to treatment or practically normal conditions around the apex.* He says, "in all cases examined, cancellous bone at the root ends of dead teeth was found to be infected;" but, as already pointed out, only one such case was cited and illustrated, the rest of his specimens being cases of eburnated bone from supposedly former dental infections.

[1] Novitzky: *Journal of Dental Research*, 1922, iv, p. 299.

To substantiate the expectations aroused by the title of this paper, Dr. Novitzky should present specimens taken from the bone around the apices of pulpless teeth which gave no, or very slight, roentgen evidence of disease.

Dr. Novitzky has not defined the term "fibrous marrow"—a term that is not commonly used in pathology. A statement regarding the intended import of this term would, I am sure, be acceptable to the readers of his paper.

I fully agree with Dr. Novitzky that new cementum deposited around a pulpless tooth does not indicate that the tooth itself is vital; that the slight amount of nutrition which may reach the root of a tooth through the cementum, as demonstrated by Dr. Box, is inadequate to maintain sufficient vitality in a tooth; and that, after extraction of a tooth, it is necessary to remove the area of diseased bone at the end of the alveolus. On the other hand, the cases cited do not give sufficient evidence to support the conclusion that *all* dead teeth are dangerous sources of infection.

All of the foregoing statements are made on general grounds. I have not had time to make microscopic studies analogous to Dr. Novitzky's, and am unable, therefore, to discuss the details of his laboratory work.

REPLY TO THOMA'S COMMENT ON NOVITZKY'S "MICROSCOPIC STUDY OF CANCELLOUS BONE AT THE ROOT ENDS OF DEAD TEETH"[1]

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When Dr. Thoma states that the conclusions drawn in my paper would lead the reader to believe that I condemn all pulpless teeth as sources of infection, he implies that all pulpless teeth should not be so condemned. This attitude might be accounted for in a mechanical dentist, following the teachings of out-of-date dental schools; but, in a lecturer on pathology, a specialist in oral surgery, it is truly startling.

Every pulpless tooth will become infected, if it remains in the jaw, no matter how it is treated. This I took as a proved fact. Further, I took it as a fact that previous investigations had shown infection in cancellous bone at the root ends of all dead teeth. The presence of fibrous marrow was described as final *corroborative* evidence. All the cases presented had histories of infected pulpless teeth. The material consisted of tissue removed by operation in my private practice during a period of some years. The examination included the bone about the root ends of many pulpless teeth in cases that were negative to roentgen-ray examination.

Special attention was given to cases that had been 'treated'—cases in which roentgen-ray examination showed what Dr. Thoma calls "improvement due to treatment, or practically normal conditions around the apex." Without a single exception, in such cases, the conditions around the apex were not normal; the cancellous bone showed infection. The roentgen-ray examination showed not *improvement* but *change in density;* and change in density does not indicate absence of infection. A few days ago I removed a large mass of fibrous granulations, most positively infected, although careful roentgen-

[1] "Novitzky's study:" *Journal of Dental Research*, 1922, iv, p. 299; "Thoma's comment:" *Ibid.*, p. 321.

ray examination had shown no difference between this mass and the normal bone.

It is true that only one of the roentgenograms presented in my paper shows the dead tooth *in situ*; but, even in this case, the microscopical examination was made after the removal of the tooth. Practical difficulties would prevent extensive microscopic examination of cancellous bone around the apices of teeth before the removal of the teeth.

Special attention was given to cases where *dead* teeth had been previously removed. This was done in order to emphasize the persistence of infection in the jaws after the extraction of dead teeth. This persistence of infection was found not only in regions of eburnated bone, located by roentgen-ray examination, but also in cases where roentgen-ray examination revealed no region of eburnation.

Such conditions as that shown in *fig. 556* may have been studied abundantly; but Dr. Thoma is undoubtedly wrong when he states that *no one* doubts that tissue taken from such a region shows an infection. Dental writers still express doubts as to the pathogenic nature of granulomata. Dental literature abounds in statements implying that infection is not always present in such regions. One dentist maintains that a granuloma is an organ beneficial to the body. Regions of osteo-myelitis are commonly pointed to as "cures," after carbolic acid or some other medicament has been poked into the septic root-canal, in spite of the fact that the very cotton that carries the medicament gives positive growth of pathogenic bacteria when later removed aseptically.

The term "fibrous marrow" was, of course, not used in dental pathology, because dentists had made no study of marrow at all. The term is used by such men as F. B. Mallory of the Harvard Medical School, and John Fraser of Edinburgh; and it is in common use at the Leland Stanford Jr. Surgical Laboratories and Hospital. The term "fibrous," I thought, would be quite clear to anyone familiar with normal lymphoid and fatty marrow.

Regarding fibrous marrow, Doctor Blaisdell writes as follows:

"The virulence of the bacterial agent and its toxin is a question of moment. It is a well known fact that different strains of the same species or variety of bacterium differ at times in their virulence. Sometimes the virulence is so great that the protective cells (fibroblasts, leucocytes,

round cells, etc.) are killed so rapidly that nature's barrier falls, and as a result the pus spreads in the direction of least resistence. Usually the toxins are moderate or even slight in virulence; and a fibroblastic activity becomes effective, and remains so as long as the injurious agent is present. In cases of latent bacteria this may extend over an indefinite period of time—even for years; a menace to be aroused by some slight trauma.

"The density of fibrous marrow is not always an index to the degree of toxicity of the injurious agents present, for the patient's individual peculiarities or health at the time determines the degree of resistance—both locally and for the system in general. In patients of low resistance, the barrier may be comparatively weak. Individual resistance varies in all degrees.

"The above facts and views are borne out by the experimental work done in our laboratories. Besides, the examination of tissues from cases of osteo-myelitis and from about the roots of dead teeth leave no room for doubt in our minds.

"Any one who has studied the inflammatory changes in bone and marrow must know that the red or lymphoid marrow and the yellow or fatty marrow become replaced by fibrous tissue in subacute and chronic affections. This fibrous tissue *is the fibrous marrow*, and usually indicates a slow chronic inflammation. It is due to a fibroblastic proliferation in response to irritation produced by the presence of an injurious agent.

"Bacterial organisms are injurious agents and their toxins constitute a group of chemical irritants. When the presence of a pus-producing organism has been discovered, either by the history of the case, or demonstrated microscopically or by culture, the cause of the presence of fibrous marrow in a case of myelitis or osteo-myelitis becomes truly evident.

"John Fraser, in his work on the 'tuberculosis of the bones and joints in children,' describes a fibro-myxomatous marrow as a quite constant phenomenon about tuberculous marrow. Here the injurious agent is the *Bacillus tuberculosis*, and its toxin constitutes a chemical irritans. In pyogenic conditions involving bone and marrow, the injurious agents are the pus-producing organisms; and their toxins, the chemical irritants.

"Fibro-myxomatous marrow is really edematous marrow, and not truly myxomatous as its name indicates. In pyogenic infections it is more truly fibrous; but, of course, it can also be edematous. We consider fibrous marrow a barrier that nature builds up to prevent the spread of infection. In the soft tissues, pus or an infected area becomes walled off by fibrous tissue to constitute an abscess; a condition that is distinctly analogous."

In conclusion, I would call attention to the nature of the comment by Dr. Thoma and to the kind of criticism that would be more valuable.

Dr. Thoma accepts my findings in each of the specific cases that I presented in support of the general statement that "our investigations showed clearly the presence of fibrous marrow in cancellous bone at the root ends of dead teeth in all cases examined." He refuses, however, to accept the general statement as the truth. He holds, on general grounds, that the cases cited do not give sufficient evidence to support the conclusion that *all* dead teeth are dangerous sources of infection. He seems to argue on the basis of theory or opinion rather than on the basis of fact. This attitude of mind is the great bugbear of the whole dead-tooth question.

When I maintained that scientific examination of thousands of dead teeth proved that all dead teeth were infected, men answered: "I do not think so," or "my opinion is different." Scientific proof, that the bone about the root end of a dead tooth was infected, was opposed by: "I think this is not true, because dental colleges do not teach it." Now, when I find that fibrous marrow, indicating infection, is present in the bone surrounding the apex of a pulpless tooth, I fully expect men to reply: "We have never heard of fibrous marrow. Hence, we do not believe you."

The questions involved are questions of fact—questions that can be answered positively and scientifically without the play of opinion. A man who has laboratories and bacteriological experts at hand can ascertain the truth or untruth of my findings. Valuable criticism would be that which would state: "I have examined dead teeth and the bone surrounding their root ends with the following results."

For years I have challenged the world to show one dead tooth, treated, or not treated, that remained uninfected in a jaw six months after pulp devitalization. Now, I suggest that a valuable kind of criticism on the foregoing paper would be that which would point to the discovery of the absence of fibrous marrow in the bone around the apex of a dead tooth.

209 Post Street

FURTHER CONSIDERATION OF SOME OF THE PROBLEMS OF TREATMENT OF PULPLESS TEETH, INCLUDING REMARKS ON RECENT CRITICISMS BY OTTOLENGUI[1]

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I. INTRODUCTION

During the period of my last year in a medical school, I devoted considerable attention to dental technics in the office of my father. The subject of pulp removal and root-canal fillings was a matter of much controversy between us. I urged him to save pulpless teeth, but he refused to leave any such teeth in the mouths of his patients. During the last year of my course in medicine, my father repeatedly called my attention to the ill health and discomforts of patients with root fillings in pulpless teeth, and their improved health after the removal of such teeth. The germ theory had just been born; and at that time (1880) I commenced experimental work on the possibility of so operating on pulpless teeth that they would not be a menace to health.

[1] The main portion of the text was read before the Pathodontia Section of the First District Dental Society of the State of New York, at the New York Academy of Medicine, April 26, 1921.

At the period of these early experiences, medicine had just passed through "the Listerian period." During an operation by the Listerian surgical technique, a number of assistants sprayed the patient, the operator, the field of operation and all his immediate environment, with solutions of phenol, in order to prevent infection. The commencement of our present-day method of aseptic surgery had just been heralded. The celebrated gynecologist, Sir Lawrence Tait, of Birmingham, England, astonished the surgical world with reports of the large percentage of recoveries in his cases of ovariotomy. As far as Tait was concerned, carbolic acid was a thing of the past; he boiled all his instruments, and used the most rigid care to obtain absolute cleanliness both in regard to the field of operation, and to his own hands and person and those of his assistants. After ceasing to use a knife or an instrument taken from the pan of boiling water, it was never re-used during the operation. In this way was born aseptic surgery of the present day. Its value was deeply impressed upon me because I was delegated by my preceptor, Dr. A. Vanderveer, of Albany, to welcome Tait to the United States, and it became my pleasant duty to assist him in many of the operations he performed here and in Albany.

My brief association with Sir Lawrence Tait gave me the impulse to attempt to handle root canals on this basis. From that time on my instruments were boiled, and creosote and carbolic acid were removed from my armamentarium, much to the pleasure of my few but sensitive patients. It must be remembered that creosote had been the foundation stone upon which all root-canal technic was based. The more foul smelling the creosote, the more favor it met from our best operators.

II. DEVELOPMENT OF A SATISFACTORY PROCEDURE FOR THE COMPLETE REMOVAL OF PULP TISSUE

From the beginning it was my object mechanically to remove all pulp tissue, place the canal in an aseptic condition, and then hermetically seal it with solid base-plate gutta-percha. In the early eighties, I introduced the solution of sublimate in Marchand's hydrogen peroxide, in order to obtain a non-septic root canal. By irrigating the canal with this solution, the free oxygen caused the loose particles

of debris to come to the surface; and I depended on the minute deposits of sublimate to keep the canal free from germs.

Shortly after the beginning of my work in this relation, William H. Atkinson and I experimented on the amputation of vital pulps. For some little time we were encouraged by seeming success, but the crop of abscesses that followed soon undeceived us. About that time (1885), Herbst, of Bremen, Germany, introduced the practice of pulp mummification, by removing the pulp from the crown only and by sealing in the canal a small amount of "cobalt."

I saw Herbst give a clinic with cobalt and my pathologic sense was outraged by it. I have never ceased to object to this form of therapy. During this period I was closely associated with William H. Atkinson, to whose farseeing pathologic sense, I feel, we have been deeply indebted. The roentgenogram had not yet been invented, and our clinics on root-canal technic, with their tedious time-taking service, found little favor with the profession, who were being more and more led astray by the exponents of pulp mummification. When the great Miller, of Berlin, already famous as a bacteriologist, wrote a paper, for *Dental Cosmos*, advocating mummification and publishing his formula for this purpose, he weakened the faith of many of our disciples.

This was the most trying period of my research in this field, because I was discouraged by the percentage of failures that I could not fail to recognize, necessitating extractions, which did not increase my popularity with my patients. It was about this period that Callahan introduced a solution of sulphuric acid for the purpose of chemically enlarging semi-calcified root canals. All the other powerful acids alone and in combination were at once experimented with. While we found that the sulphuric acid was of the greatest value in obtaining better results, it fell short of solving the problem of removing every vestige of pulp tissue. It failed utterly to destroy the inaccessible pulp tissue that was free from calcification. in fact, it only brought more adherence to the practice of mummification.

Greatly discouraged at being so near to, yet so far from, the goal of removing every particle of pulp tissue, I attended the First International Dental Congress, held in Chicago in 1893, where I was destined to find the chemical agents which have enabled us to over-

come that greatest of obstacles—the removal of pulp tissue from inaccessible canals and ramifying branches.

During the sessions of the Congress, Emil Schreier, of Vienna, gave continuous clinics with his mixture of sodium and potassium (kalium-natrium). It was the most popular clinic at that great meeting of dentists. He introduced, and sealed in the root canals, a mass of his amalgam compound in the same way in which all the mummifying preparations were being used. It was supposed to be the latest formula for pulp mummification. He had invented a method of combining these two metals, sodium and potassium.

I watched Schreier's first clinic with a feeling of skepticism, but was at once fascinated by the same thing that attracted everyone. When this amalgam-like mixture came in contact with pulp tissue, there was generally an explosion, frequently a flame, much gas, but nothing remained of that particular portion of pulp tissue. Again and again I returned to his clinic to watch this destruction of pulp tissue, and realized that the agent had been finally invented by a dentist which could remove every vestige of pulp tissue contained within the root canals. Schreier visited me on his way through New York, and then I demonstrated to him in my office that its real value consisted in his capability of destroying every vestige of organic substance.

Sodium-potassium should be introduced in the smallest quantity, in order to destroy a limited amount of tissue at a time. Used in large masses there is always danger of destroying organic cement substance in the dentine itself. The destructive power of this agent is very much increased by the intense heat resulting from the chemical union of the water in the organic substance with the sodium-potassium. There is an entire absence of escharotic action such as results when sulphuric acid is used under similar conditions.

After its escharotic action has been accomplished, sulphuric acid leaves an inorganic precipitate, which cannot be absorbed. While this result is harmless in the canals, it is not a satisfying reaction for the periapical region.

III. ON THE NECESSITY FOR PASSING THE PULP-REMOVING MATERIAL THROUGH THE FORAMEN

To obtain the maximum benefit from the use of sodium-potassium, the material should be passed through the foramen. No one should hesitate to use it in this way, even in cases of removal of vital pulps. The ultimate junction of pulp tissue, at the foramen, with periapical tissue, has often been spoken of as the physiologic encapsulation of the root end. My clinical records show many cases where this small amount of living tissue soon died and became the initial factor in a focal infection. The fact that this small amount of pulp tissue may retain its vitality for variable periods of time, in some cases, does not warrant its retention in any instance, when we consider that many cases of infection arise solely from this reaction to lack of operative thoroughness. There are, of course, exceptions to this rule of practice. W. Clyde Davis has recently called our attention to a series of pulpless teeth where new cementum has sealed the foraminal entrances and extends some distance up the canal. These results undoubtedly are found in only a small proportion of teeth. If the diagnosis of this condition can be made, there ought to be no necessity for attempting to go further than the root filling of cementum, as this leaves an ideal encapsulation if the new cementum hermetically seals the foramina. This diagnosis is not easily made, but it will undoubtedly be frequently given as an excuse for imperfect and slovenly work. Incidentally, it might be stated that the cases reported by Davis afford an additional argument against the absurdity of regarding a pulpless tooth as *necessarily* a dead tooth.[2]

About eighteen months ago, in collaboration with Drs. Gies and Bastian, we injected trypan blue into two dogs. These injections

[2] Since the above was written, Davis has withdrawn his statement on the possibility that cementum can fill the dental foramen, and assumes that, in the cases he has had under observation, calcification is in progress, which he calls "osteoid." This simply expresses an observation, on his part, of the ordinary calcific degeneration that occurs in so large a percentage of root canals. Careful observation on my part, covering a period of over forty years, has shown that this calcific matter is intermingled here and there with organic layers, which easily become seats of infection. It also demonstrates the lack of dependence that can be placed upon such hasty and careless observations as those of Davis. His work in this connection is simply another effort in the field of embalming (Egyptian dentistry!) No words of warning can be too strong against the profession's giving serious consideration to doctrines such as these.

were repeated at intervals until we established a well marked blue coloration of each dog. The pulp of two of the incisors in each of these dogs had previously been removed and good gutta-percha root fillings inserted. At a later period, after these dogs had been chloroformed, the jaws were very carefully examined. It was observed that the trypan blue had found its way into all of the tissues, hard and soft, including the bones and the teeth. Individual teeth were removed from the jaw, split, and examined carefully. The periodontal membrane in each case was found to be thoroughly impregnated by the pigment, and the teeth themselves assumed a distinctly bluish tint. As our research in this direction is to continue, I prefer to postpone discussion of this comparison. One finding was impressive. Although the periodontal membrane around the apex was completely discolored by the trypan blue, none of this pigment passed through the foramen into the canal. The pink gutta-percha root filling failed to show a trace of discoloration. This must be considered good evidence that base-plate gutta-percha and chloroform will hermetically seal root canals.

In an investigation I reported to this Section over a year ago, the apical thirds of some teeth were covered with gutta-percha, chloroform and rosin; others, with chloroform and rosin; and others, with chloroform and gutta-percha. These specimens were tightly sealed, some in vials of distilled water and some in physiologic salt solution. Within thirty-six hours the water in the vials holding any mixture of rosin contained numerous organisms that continued rapidly to multiply. The water in the vials containing teeth covered with gutta-percha and chloroform remained unchanged during weeks of observation. I also called attention to the fact that rosin and chloroform fail to yield a perfect mixture. Their mechanical mixture is a mass filled with air bubbles, which tend to make the mixture exceedingly brittle when it finally hardens. This condition has accounted for the fact that a great many encapsulations have broken away from the end of the root, and it is very timely to have it understood that gutta-percha and rosin root-fillings should never be confounded with a correct base-plate gutta-percha root filling. The latter material never changes its physical characteristics after it has once been placed in position. All rosins contain numerous varieties

of organisms and consequently the use of gutta-percha and rosin must be condemned.

The use of sodium-potassium, as above described, has been severely criticized by different men. This criticism has been mainly directed against its destruction of tissue in the periapical region. From a general dental standpoint, Grove and Grieves appear to have made out a very strong case against its use. Let us consider, for a moment, the bugaboo of periapical destruction caused by the use of sodium-potassium. It destroys tissue, diseased and healthy, and its use can be appropriately compared to that of the surgeon's knife. Good surgery demands not only the removal of the diseased mass, but also that of surrounding healthy tissue. The surgeon does this fearlessly because of the recognized regenerative power of all tissue, unless infective forces are present. To criticize a surgeon for removing healthy tissue in the environment of a diseased mass would receive no serious attention in any surgical assemblage. In our therapy, sodium-potassium takes the place of the knife, a fact that should be a sufficient reply to criticism of this nature.

When a root canal is opened by utilizing the chemical action of sodium-potassium, by the very virtue of its action as it advances toward the foramen of the tooth, infection is automatically eliminated. Its own destructive action makes it impossible to force infected pulp through a foramen. Blocking the nerve trunk with novocain, as a local anesthetic, generally renders the use of sodium-potassium entirely painless, whenever this is found to be required.

Grieves is deserving of great praise for his excellent classification of pulpily and apically diseased teeth. There is no reason why this classification should not be generally accepted. His conclusions, however, as to treatment, are wholly at variance with my own views, which are based entirely on clinical data that can be best expressed as follows: All pulp tissue having been removed and periapical disease, if existent, eradicated, successful root-canal therapy is in the last analysis dependent on ideal root encapsulation. The basis of this claim rests on evidence from roentgenograms taken year after year after such encapsulation.

At the very commencement of my practice, I began to look to surgery for aid in removing all diseased periapical tissue, and soon

was amputating root ends as a regular routine of practice. I presented this operation before the American Dental Association at Excelsior Springs in 1890. Where scientific root-canal treatment has been used, there should be very little difficulty in surgical removal of infected tissue. The postoperative danger in these cases lurks in a possible osteoclasia, resulting in destruction of the root end.

In some recent research, Hopewell-Smith has shown that, besides the danger of osteoclasia, root ends are subject to caries. This undoubtedly shows the presence of bacteria deeply lodged in the alveolar structure. My clinical observation, for over forty years, long ago led me to conclude that presence of a great amount of infected tissue is the cause of operative failure in many cases. The many postmortems that have shown heart muscle to be thoroughly impregnated with streptococci suggest how these organisms may, in like manner, be thoroughly imbedded around the apical region of a pulpless tooth; this tissue being for the time the *locus minoris resistentiae*. On this account, the destruction or eradication of a limited amount of infected periapical tissue is insufficient, in a percentage of cases, to restore healthiness to teeth. The proper therapy must protect exposed cementum or dentin from such attacks. The use of sodium-potassium at once produced a large decrease in the number of cases that called for apicoecotomy.

During the last year, Levene, of the Rockefeller Institute, suggested sodium methylate as a substitute for sodium-potassium, with the hope that it would be less irritating. Chayes has been the first to use it in dental practice. Its use, in my hands, has been less effective than that of sodium-potassium. Unfortunately, in my experience, there has not been less irritation. In the cases where I have used it, there has been irritation to a very marked extent. With novocain at our service, pain can be very largely eliminated. The important factor is use of the compound that will most effectively ream our canals and obliterate the organic tissue. For this purpose sodium-potassium appears to be the most resourceful compound at our command.

IV. THE REMOVAL OF ALL DISEASED PERIAPICAL TISSUE IS ESSENTIAL; THE UTILITY OF ELECTROLYTIC TREATMENT FOR THIS PURPOSE

The second requisite in root-canal therapy is the removal of all diseased tissue situated in the periapical region. This undoubtedly is most easily and thoroughly accomplished by surgical procedure. Unless this includes, however, the clean amputation of the end of the root, there is grave danger of destroying the periodontal lining at the end of the root, which invariably means re-infection. The danger of root absorption resulting from osteoclasia and caries is present in all forms of surgical procedure. On this account it was very desirable to find other means for the obliteration of pathologic conditions in the periapical region.

The utility of the galvanic current for cataphoric anesthesia very soon led me to employ it for the purpose of electrolytic medication. Since 1894 I have been using the current for this purpose. From a clinical standpoint I was soon impressed by the wonderful therapeutic value of the current that carried any desired ion into the periapical region. The number of resultant cures, and of teeth that were maintained free from re-infection, was certainly impressive. While its use is very largely empirical, clinical successes, repeated again and again, cannot be overlooked, and it only remains for intensive investigation to afford an explanation of the manner in which the curative results are obtained. This form of ionic medication requires an unending amount of patience and perseverence on the part of the dentist. At the very outset, in order to make the application painless, considerable time must be taken in the most careful gradation of the current until the largest possible amperage passes through the tooth without discomfort to the patient. Many of our best operators have failed in the use of electrolytic medication because of failure to force a sufficient amount of ions through the foramen. At the beginning, in special cases, cultures were taken at intervals from the periapical region; and the fact was noted that, in a large number of cases, no growth was obtained when these cultures were taken immediately after electrolization. When, however, the patient returned in a week, new cultures generally again yielded growths. This persistent clinical fact led me to use a continual increase in the degree of ionization in a given case.

Three years ago, in collaboration with Drs. Gies and Levy, I began a compilation of statistics on this form of treatment. Since then every one of my cases of pulpless teeth has been subjected to careful culturing. This paper is not intended to be even a partial report on that research. I feel, however, that I have data enough to justify certain conclusions on the subject of electrolytic medication, the benefit of which should not be longer kept from the profession. Up to date I have sent to the Columbia University laboratory over forty-five hundred tubes to be cultured. Two remarkable facts have been noted. *First:* given a well defined region of infection, cultures taken from this region, immediately after thorough application of electrolytic medication, show no growth in over 90 per cent of the cases, although, in over 60 per cent of these cases, a second culture, taken a week later, yields a growth of streptococcus. These facts led to long continued use of constantly increasing quantities of current, often extending over a period of months, before filling the root canals. In many of these cases where the treatment extended over weeks, the total milli-amperage of applied current frequently amounted to as much as eight to ten milli-ampere hours. All of these cases have been kept under careful roentgenographic observation. This, in turn, has brought to light the *second* fact: wherever ideal encapsulation was possible, it was followed by ideal regeneration.

In an article published by Grieves, in the April (1921) number of the *Journal of the National Dental Association*, he lays great stress on the importance of the quality of roentgenograms, and the danger of confounding pathologic conditions with alveolar regeneration. He is very skeptical about alveolar regeneration. While admitting that it does sometimes occur, he says: "The trabeculae and partitions of the adjacent normal bone to which these should be compared are missing." The pictures of regenerated bone that I shall have the pleasure of showing you display all the trabeculae and partitions. I desire especially to call your attention to the gradual improvement, from a histologic viewpoint, of these tissues as time goes on. As the encapsulation becomes a year older, the markings of the trabeculae and partitions of the regenerated tissue become better defined. I feel, further, that I cannot agree with his statement that "apical rarefaction is diagnostic of a dead apex." The destruction of the

alveolar structure by no means implies necessary damage to the periodontal lining; and as long as this membrane remains intact, with a sufficient circulation, it is most inappropriate to speak of the end of the root as being dead. There is no doubt that in some cases there is necrosis of the apex of the root. I have no knowledge, however, of any such case where it was not accompanied by suppuration of a character that always attends necrosis of bone or tooth structure. Such teeth should not be retained; but, on the other hand, they should not be confounded with teeth on which the pericementum surrounding the roots has been entirely destroyed.

V. A PROPORTION OF FAILURES IS INEVITABLE IN EVERY SURGICAL PROCEDURE

A certain percentage of failures occur in these operations as they do in all operations on the living body. Whenever the roentgenogram shows a failure in regeneration of the alveolar structure, it stamps the operation as a failure, and recourse must then be had to apicoectomy. Failing there, extraction of the tooth and currettement of the process are imperative. I shall show you pictures of successful operations. I shall show you, also, some cases of failures. In these failures we are confronted by the fact that the patient generally has no discomfort. Patients generally fail to have a proper understanding of the desirability of immediate removal of the tooth, etc. It is always necessary to make the patient thoroughly familiar with the reasons for a failure and with the danger of retaining such a tooth. Again, it demands the highest order of moral courage to acknowledge your failure when you know that, if you kept silent. the patient would never be any wiser. By admitting failure you run the risk of losing your patient, who in some instances seems to be unable to appreciate the virtue of your attitude. The *crime* of withholding such information from your patient, however, is obvious. In every other field of surgery, a percentage of failures is freely acknowledged. The same allowance will be made for dental surgery, when dentists freely admit, as they should, that there is a percentage of unavoidable failures.

VI. REMARKS ON SOME OF OTTOLENGUI'S RECENT CRITICISMS

The June (1922) number of *Dental Items of Interest* publishes my discussion of a paper by Ottolengui on this subject. During that discussion I displayed a few of the roentgenograms reproduced in this paper. Ottolengui has seen fit to criticise my discussion in the same issue. If he had sent me a copy of his comment, I would have gladly replied so that my response could have been printed in the number showing the illustrations. Comment where the illustrations would not be available would be useless. On that account, it seems desirable here to answer Ottolengui's criticisms, in the description of my illustrations. (See p. 341.) Let not the members of the "100-per cent club" jump to the conclusion that, because we differ in some respects, we are engaged in a deadly feud. We are in thorough harmony on the important fact that a certain percentage of pulpless teeth can be maintained in the jaw when properly treated, without becoming a menace to the individual's health. Personally I feel, with Ottolengui, that these differences of opinion should be freely aired, because they involve very vital principles in certain classes of cases. We are all liable to error. If I am in error, I trust I may live long enough to admit it. I believe the correctness of my view will be recognized by the dentists of the future, who will be as thoroughly educated in every branch of medicine as every other specialist in the healing art is supposed to be.

While Ottolengui at heart has no fault to find with my treatment of infected teeth, he and many of my friends are shocked at my utter disregard for the maintenance of what they call the "physiologic encapsulation" in recently devitalized teeth. It is certainly true that many teeth made pulpless by trauma of some kind, and many teeth with imperfect root fillings, have been under years of observation without showing a trace of infection. This condition of affairs is freely acknowledged even by indifferent observers. For this very reason, the lack of a perfect operation does not necessarily spell toxemia, and many able men fail to discover the flaws in their technic.

VII. THE IMPORTANCE OF ENCAPSULATION OF ALL FORAMINAL OPENINGS

At the beginning of this paper I referred to the unsatisfactory experiment, in the '80s by Atkinson and myself, in pulp amputation.

We left a large amount of pulp tissue in some cases and very little in others; and, though working under aseptic auspices, Atkinson became convinced that the procedure was wrong. William H. Atkinson was the greatest dentist I ever met. Having once come to the conclusion that all of the pulp tissue must be removed, he at once acted accordingly. With a large surgical drill he would go straight down the canal (when feasible) and straight through the foramen, never stopping until the blood flowed into the canal. Then followed intense medication of the periapical region, with the most powerful escharotics to take the place of the knife. He was the first to show me the use of gold for root-canal fillings; but, at the time, he said: "we must find an ideal root filling and it cannot be a cement." Those were the days of the dental giant. I will never forget the first clinic in New York by the beloved McKellops, of St. Louis, in which he filled a root canal with gutta-percha (and used chloroform). When it was over Atkinson exclaimed, "eureka," and gave me such a hug that my ribs were sore for days. Those were the days when oxychloride-of-zinc cement was used for root-canal fillings. We fought the cement root-canal filling at every point because we knew it was wrong.

In voicing his opposition to deliberate insulation of the end of a root, Ottolengui asks: "is there any analogous treatment in the entire realm of surgery?" Teeth do not normally grow in any other place than the alveolar process. Into this process come microörganisms hunting for sustenance, which is most abundant at the end of the root. How much of the alveolar tissue is infected by such microörganisms no one can tell. It is a common error to fail to realize the great extent of the infected region. It is usually forgotten that when a few million germs in the front ranks are destroyed, other millions at once step forward to take their places. These germs are looking for sustenance, which can always be found around any foraminal opening. It is this use, by pathogenic germs, of the tooth as a storehouse for food that makes an infected tooth a menace to health. It is impossible to determine that no more germs will *visit* this region, but, *obtaining no food there*, they die rapidly, and are harmless.

The main body of a pulpless root is united by the periodontal membrane to the periosteum of the jaw. Only the root tip is without a

vital connection. This tip may be just the foraminal canal or it may be a considerable portion of the root end. It is composed of inorganic and organic materials, all capable of retrograde metamorphosis, if it comes in contact with vital tissues. Here is where the wonderful value of insulating this most vulnerable part of the tooth comes in. Base-plate gutta-percha, an inert substance and non-irritating, is utilized to encapsulate this exposed root end. It is true that the encapsulation must be perfect in order to leave the tooth ideally defended. After hundreds of cases have proven the correctness of this therapy, it remains no longer a theory, but a fact. Skepticism and ignorant criticism, however powerfully expressed, cannot remove the roentgenographic record of successes when encapsulation was successfully accomplished. Even the record of observed failures, through inability to make encapsulation a success, becomes a proof of the soundness of this therapy.

If this point is well taken, then it should require little argument to convert dentists to the use of the encapsulation therapy for every pulpless tooth. If no infection has ever been present, the more the need for encapsulation as a great prophylactic measure. It is here that the greatest objection arises to the use of rosin as advocated by Ottolengui. While base-plate gutta-percha is an inert substance, rosin has been found to be filled with organisms and consequently is absolutely unfitted for insulation purposes. In the technic advocated by Ottolengui, the rosin solution under ordinary circumstances must go through the foramen, if the filling is to go anywhere near the end of the foramen. It is easy to see that this material will not do for insulation purposes.

In his criticism of my discussion, Ottolengui makes a great point of showing the air spaces where chloro-percha is dropped on a slab and allowed to harden. No one contradicts the truth of this; in fact we might add that these "*igloos*" are twice as large when, according to Callahan, rosin is added to the solution. Ottolengui realizes that I shall vigorously answer this statement by saying that we do not fill root canals with chloro-percha, and he attempts to answer my answer before he has heard it. We get rid of these air spaces by long continuous "mortarization" of our gutta-percha. The canal is filled with chloroform into which we introduce our gutta-percha points. The

canal is the mortar; the plugger is the pestle, which turns around and around the macerated gutta-percha, making pressure uniform against every possible space that the gutta-percha enters. This "mortarization," kept up long enough, gives us a homogeneous mass in every canal space and so compresses the gutta-percha that all spaces disappear. The fact that these root fillings inhibit the passage of any colored fluid should be a sufficient reply to every criticism in this relation. In fact, at the close of Ottolengui's criticism of encapsulation we are met by this paradoxical sentence: "*I have little doubt that a homogeneous gutta-percha root filling, inserted with Rhein's technique, will inhibit the entrance of bacteria.*" As this is the goal of all our root-canal therapy, why the objection?

Unfortunately, many men fail to obtain a true picture of the technique of my operation from reading my articles. It is impossible for me to express in words the delicacy of manipulation, the length of time, and the unflagging patience required of the operator. Only a practical training at the side of the patient, or at the technique table, can make this properly understood. Consequently, I beg my colleagues not to condemn my operation before they have personally seen the operation *properly* performed.

In the lantern slides I am about to present, I show all kinds of cases—successes and failures—those of real instructive benefit. It shall be my endeavor to reply to Ottolengui's criticisms of them in the appended descriptions.

VIII. DISCUSSION OF THE ILLUSTRATIONS (*figs. 1–24*); AND FURTHER COMMENT ON CRITICISMS BY OTTOLENGUI

Fig. 1. Shows a gutta-percha root filling, inserted nearly thirty-two years ago in the upper canine of a young girl, the pulp having been traumatically devitalized. A few years ago the patient, now a mother of quite a family, presented with a traumatic devitalization of the adjoining first bicuspid. The roentgenogram shows a large radiolucent periapical area. Nevertheless, there is no gainsaying the absolute normality, histologically, of the alveolus in which the point of the gutta-percha cone used in the root filling of the canine is protruding. This root filling was not affected by a very bad automobile accident.

Ottolengui asks: "What does this prove?" It proves conclusively that, notwithstanding the protrusion of the cone of gutta-percha, if everything was aseptic and if the gutta-percha point hermetically sealed the foramen in passing through it, the result must be a return to physiologic tissue. Ottolengui asks: "If the root filling had stopped exactly at the foramen margin, would not the result have been as good?" The answer depends entirely on what Ottolengui means by "foramen margin." If the filling failed, by any unmeasurable length, to seal the outer margin of the foramen. it would not only not have been as good, but it would have been a total failure in preventing bacteria from obtaining sustenance in the crater-like beginning of every foramen. The filling must cover the outer lips of this crater-like opening in order to prevent the entrance of germs.

Figs. 2 and 3. Practically the same kind of case as that in *fig. 1* is shown. Although the adjoining first bicuspid was extracted because of an incurable infection, it failed to interfere with the ideal regeneration of the alveolus under the second lower bicuspid.

Fig. 4. A roentgenogram of an upper pulpless lateral showing the diagnostic wire protruding. (See *fig. 5.*)

Fig. 5. A roentgenogram of the tooth shown in *fig. 4*, after the root canal had been filled. This is a remarkably interesting and instructive case, and yet Ottolengui failed entirely to appreciate why it had been introduced. In his reply, he claims to match it with a root filling in a lower tooth which apparently shows encapsulation. While the encapsulation in *fig. 5* is exceptionally good, it was not introduced to show that fact. *Fig. 5* was introduced to show that there was no unusual amount of reaming. The filling, however, shows all kinds of accessory openings leading to the pericementum. Two of these canals are very large in diameter; others not so large. The picture is given in order to demonstrate that while we have an ideal encapsulation of the end, the gutta-percha did not pass through this foramen beyond the encapsulation. It did, however, ideally fill all these subsidiary canals and encapsulate them at the same time. The picture is intended to show how "mortarization" forces the macerated gutta-percha into every opening, of which there were many.

Figs. 6 and 7. These roentgenograms show a lower-bicuspid root canal during the process of root filling. The end of the root shows

FIG. 1. ROENTGENOGRAM OF A PROTRUDING GUTTA-PERCHA ROOT FILLING; INSERTED NEARLY THIRTY-TWO YEARS AGO IN AN UPPER CANINE OF A YOUNG GIRL, THE PULP HAVING BEEN TRAUMATICALLY DEVITALIZED

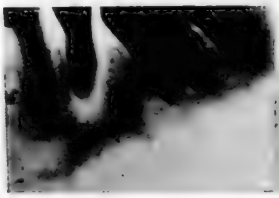

FIG. 2

FIG. 3

FIGS. 2 AND 3. ROENTGENOGRAMS OF A CASE SIMILAR TO THAT SHOWN IN FIG. 1

The figure at the left (2) shows a granuloma at the end of a lower second bicuspid; November 4, 1911. The figure at the right (3) shows *ideal regeneration*, nine years and three months later; February 11, 1922.

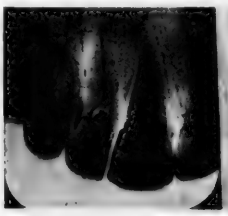

FIG. 4

FIG. 5

FIGS. 4 AND 5. IDEAL FILLING OF THE SUBSIDIARY CANALS AND PERFECT ENCAPSULATION WITHOUT UNNECESSARY PROTRUSION

The figure at the left (4) shows an upper pulpless lateral with a diagnostic wire protruding. The figure at the right (5) shows the condition of the tooth after the root canal had been filled.

that an absorption process had been started, exposing some of the cementum, bringing the case under a classification where the percentage of successes is very low because of the necessity of obtaining absolute encapsulation over all exposed cementum. The first picture is taken with a rubber dam in position, and shows what may be termed a filling to the end of the canal. According to Ottolengui, I should have stopped there and considered the operation ideal, even though exposed cementum is shown. On the contrary, "mortarization" of the gutta-percha was continued until (*fig. 7*) I obtained ideal encapsulation not only of the foramen, but also of the end of the root, including the exposed cementum. It is difficult to understand why Ottolengui should fail to concede that an ideal result was produced in this typical case.

Figs. 8 and 9. These roentgenograms show a similar value of the "mortarization" technic, carried to its ultimate conclusion. *Fig. 8* shows a lower molar where an unusual amount of gutta-percha was packed into the mesial canals. With a feeling of confidence the rubber dam was removed, but our disappointment was great to find, from our roentgenogram, that gutta-percha was quite short of the end of the root. Two days later the gutta-percha was re-softened with chloroform and "mortarization" kept up until the beautiful encapsulation shown in *fig. 9* was obtained. Note that this time the picture was taken *before* the rubber dam had been removed.

Fig. 10. The first of these five roentgenograms of an upper bicuspid was taken in January, 1912; the last, *in San Francisco, on August 10, 1922.* The case has always been the most instructive in my case books, because it clearly proves that (a) the etiologic factors in a case of acute arthritis were the toxins from a traumatically devitalized pulp; that (b) removal of the pulp and insertion of the root filling cured the arthritis; and that (c) there was a steady improvement in the histological character of the regenerated alveolus. The story is well worth recording. Miss K., age 18 (my patient for many years, with very few fillings and an ideally sanitary mouth), in a storm while crossing the ocean to Italy, was thrown from her berth, and her head badly bruised and face disfigured. There was no suspicion of any injury to her teeth. Two weeks after her arrival in Rome a knee commenced to swell, and she was compelled to use crutches for the

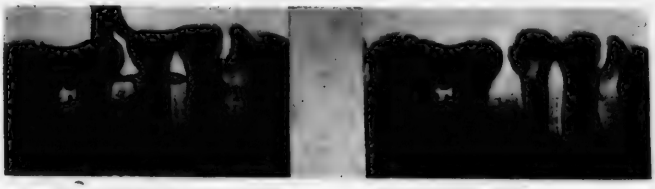

FIG. 6 FIG. 7

FIGS. 6 AND 7. IDEAL ENCAPSULATION, AFTER "MORTARIZATION"

The figure at the left (6) shows a lower bicuspid root-canal during the process of root filling; apical absorption had been under way and cementum was exposed. The figure to the right (7) shows ideal encapsulation, not only of the foramen but also at the end of the root, after "mortarization."

FIG. 8 FIG. 9

FIGS. 8 AND 9. PERFECT ENCAPSULATION, AFTER RE-TREATMENT

The figure at the left (8) shows a lower molar with an unusual amount of gutta-percha packed into the mesial canals. The figure at the right (9) shows beautiful encapsulation. after re-treatment, including softening of the gutta-percha and "mortarization" of it.

FIG. 10. STEADY IMPROVEMENT IN THE CHARACTER OF THE REGENERATED ALVEOLUS

Roentgenograms taken successively (left to right) in January, 1912; May, 1912; February, 1919; December, 1920; August, 1922.

year and three-quarters that she remained in Italy. After her return to the United States, she was under the care of a skilled orthopedist, but he was unable to benefit her. About six months after her return, she felt for a few moments a slight irritation over the root of the second bicuspid. A roentgenogram gave us the first indication of the presence of a well marked granuloma. The tooth had no filling in it and was without any defect. Under strict asepsis the pulp chamber was entered and found to be filled with only a brownish fluid. This was siphoned into an aseptic tube and cultured. No growth could be detected before the ninth day. The organism was a typical streptococcus viridans. Zinc electrolytic treatment was given and the root canal filled. The patient was able to discard the crutches after the first week, and up to date there has been no recurrence of arthritis. The pictures show the gradual disappearance of the granuloma and complete osseous regeneration.

In the second picture, in *fig. 10*, there is a slight excess of protrusion of gutta-percha. As stated previously, there is always a possibility that such a protrusion will break off; it did so in this case. This is not an unusual occurrence, and invariably the small particles of gutta-percha are encysted in the regenerated alveolar structure. This small particle of gutta-percha can be found slightly to the right of the tip of the root. The reproduction of these pictures (first four), in *Dental Items of Interest*, was so poor that this particle can scarcely be found in them, but even then it can be detected. Ottolengui very disinterestedly hints that the particle might have been exfoliated through a fistula. There has never been a fistulous opening in this case, nor the slightest local disturbance. It might as well be said, here and now, that Ottolengui has only a theoretical understanding of my technic. He has never had any practical instruction in my method. When he assumes the possibility of a secondary infection, he shows a blissful ignorance of how we operate, and illustrates in a practical way the truth of my unhappy conclusion that it is well nigh impossible for any one to appreciate my technic who does not watch it in practice.

I have treated many cases of infection that were so bad that I found them incurable. I have a file of all the original records of my cases dating back to May, 1881. It is impossible to find a case

in my records, handled by *myself*, where, after a root canal has been filled, any infection ever started that would naturally result in a fistulous opening. My claim is that, with a proper understanding and execution of aseptic procedure, such a result (very common, I am given to understand) would be impossible. This is no idle boast; and any visiting committee can have the opportunity, at any time, of checking up this statement.

Fig. 11. These roentgenograms show one of the most beautiful cases of regenerated alveolar structure on record. A girl, age 17, had the two lower incisors traumatically devitalized in a basketball game. Treated at first by her dentist, in Halifax, who found himself very much perplexed, especially as a large mass of gutta-percha had been forced through the foramen, she was sent to me on November 21, 1919, when we took the first picture. The second picture was sent to me from Halifax by her dentist, and I could not control the angle at which it was taken. The lump of gutta-percha is just discernible at the bottom of the first picture. This difference in the angle of the picture taken by me, and the one taken by her dentist after regeneration was complete, has again prompted Ottolengui to question the accuracy of my statement that all the bone between the two incisors was missing when she came to me. My statement would not have been so positive if there had been any uncertainty; and the history of the treatment, studied with the aid of the accompanying roentgenograms (*figs. 12, 13, 14 and 15*), is most positive proof of the correctness of my statement.

Figs. 12, 13 and 14. These nine roentgenograms were taken successively on November 21 and December 10, 1919; January 7, 12, 17, 22 and 28, February 3, and March 10, 1920. The very first picture that was taken (*fig. 11*) showed the dislodged mass of gutta-percha in the alveolar space far below the ends of the roots. On the same day, after treatment of one of the teeth, the lump of gutta-percha is shown (in the first picture in *fig. 12*) to be between the roots, halfway to the crown. All of the succeeding pictures show it in different positions, which means that there was a large empty space between the roots and below them. Electrolytic medication in an intensive manner was administered on the six dates specified for *figs. 12 and 13;* but, up to January 22, 1920, it was impossible to remove the floating piece of gutta-percha.

FIG. 11

FIG. 12

FIG. 13

FIG. 14

FIG. 15

FIGS. 11, 12, 13, 14 AND 15. A CASE INVOLVING TRAUMATIC DEATH OF PULPS; IMPERFECT ROOT THERAPY; A MASS OF GUTTA-PERCHA IN THE PERIAPICAL REGION UNDER TREATMENT; REMOVAL OF THE LATTER; COMPLETE REGENERATION

Fig. 11. Shows complete regeneration of alveolar structure. The roentgenogram at the left was taken in November, 1919; that on the right, in January, 1921. See *fig. 15.*

Fig. 12. Therapy and efforts to remove the particle of gutta-percha continued. The roentgenograms were taken (left to right) on November 21 and December 10, 1919 and on January 7, 1920.

Fig. 13. Therapy and efforts to remove the particle of gutta-percha continued. The roentgenograms were taken (left to right) on January 12, 17 and 22, 1920.

Fig. 14. Therapy continued and the particle of gutta-percha removed. The roentgenograms were taken (left to right) on January 28, February 3, and March 10, 1920.

Fig. 15. Same as the right half of *fig. 11*; taken in January, 1921.

On January 28, 1920, the bacteriologic condition had improved to such an extent that we completed both root fillings, made a very large incision between the incisors, and removed the floating piece of gutta-percha. In *fig. 14*, you will note (February 3) the results of a rapid regenerative process then already in progress. On March 10, this regeneration had advanced much farther. The patient then returned to Halifax.

Fig. 15. This roentgenogram was sent to me from Halifax the following January (1921). The remarkably rapid physiologic regeneration was largely due to the youth of the patient, after all infection had been removed. At the same time, I am convinced that the intensive electrolytic treatment exercised a marked beneficial action on this ideal return to normality.

Some of our cases do not turn out successfully. I shall show some of the failures, in order that it may be seen that we do not claim 100-per cent success; and to show, also, that we learn what we can from our failures.

Fig. 16. These roentgenograms were taken from a young woman who was a patient in one of our classes. On December 6, 1918, we started operations. The infected region is well defined. At the next meeting of the class, in January, 1919, the root canal was filled, apparently very successfully. The patient had the root crowned by another dentist. The following December (1919) as the third picture shows, there was marked reinfection. Personally I place the blame for this failure on insufficiency in the amount of applied electrolytic medication. The class wanted to see the root filled, and this was done before a favorable bacteriologic condition was obtained. The lesson taught by this failure was impressive.

Fig. 17. A successful result is shown. (See *fig. 18.*)

Fig. 18. These roentgenograms show the bicuspid on the opposite side of the mouth of the patient involved in *fig. 17*. Here again we have a marked failure due, very likely, to a desire to fill the root canal before our bacteriologic condition had sufficiently improved. Both these teeth were extracted. Cases like these have convinced me that, if electrolytic treatment is continued far enough, its possibilities are unlimited.

Fig. 19. This roentgenogram was taken from a woman, aged over 50, who had been a severe sufferer from arthritis; and also had been

FIG. 16. A FAILURE; REINFECTION

The roentgenograms were taken (left to right) in December, 1918; January, 1919; and December, 1920.

FIG. 17. A SUCCESS

The roentgenograms were taken (left) on February 20, 1919, and (right) June 19, 1919.

FIG. 18. A FAILURE

The roentgenograms were taken (left to right) on January 24 and 31, and April 4, 1919.

FIG. 19 FIG. 20

FIGS. 19 AND 20. A SUCCESS

The roentgenograms were taken (19) in 1917 and (20) 1919.

suffering from tic in the right incisor fossa. The right lateral, left central, and lateral, were pulpless; roentgenograms showed large radiolucent areas. The right central contained a living pulp that was partially calcified and causing continuous pain. This living pulp, as well as the defective root fillings in the other three teeth, were removed. *Fig. 19* shows the result immediately after the operations. The patient resided in Georgia and I referred her to Dr. Hinman, of Atlanta, as she required other treatment. She arrived in Atlanta at the time of the annual dental meeting there, in 1917, and was inspected by Drs. Hartzell and Grieves who happened to be there. They were insistent on curing this case at their clinic by amputating the root ends of all four teeth. Dr. Hinman's associate, Dr. Johnston, however, recognized the success of my operation; and the patient, following his advice, refused to be operated upon.

Fig. 20 shows the conditions two years later, ideal regeneration having taken place. Incidentally, every symptom of tic and arthritis disappeared. She has thrown away her cane and is an indefatigable pedestrian. This case teaches us the necessity of being careful not to make a hasty diagnosis. How many of us require this lesson will be left for posterity to determine.

I am reserving my best case for the last. It has been stated by many authorities that where absorption of the root has taken place, so that cementum is exposed, it is useless to endeavor to treat such a case. (See *figs. 21–24.*)

Fig. 21 shows a lower bicuspid. It must have been pulpless for years, without giving trouble or causing any suspicion of its condition, when, during an intra-oral inspection, this condition revealed itself. The prognosis was so unfavorable that I advised extraction. I was asked to see what could be done, however, and at once determined to use electrolysis "to the limit." The granuloma at that time was well marked. After sufficient treatment had been given, the one hope was to have the gutta-percha thoroughly encapsulate the absorbed root end. How thoroughly we succeeded is revealed in *fig. 21*, which shows on the left side the granuloma, etc., and on the right side, over a year later, the thoroughly encapsulated root end, with ideal alveolar regeneration. I am convinced that this wonderful result would never have been attained if it had not been for the long

continued use of the galvanic current. From February 12 to April 30, 1920, I applied over 25 milliampere hours of current, with iodine solution. It was only after repeated bacteriologic tests had failed to yield growths that, on April 30, 1920, the root canal was effectively filled. A few of the many roentgenograms taken during this treatment are shown in *figs. 22–24.*

Fig. 22. A roentgenogram taken on March 13, 1920, after six different treatments; shows very little change. The cured condition is seen in the second roentgenogram, taken, at the same angle, on November 23, 1920.

FIG. 21

FIG. 22

FIG. 23

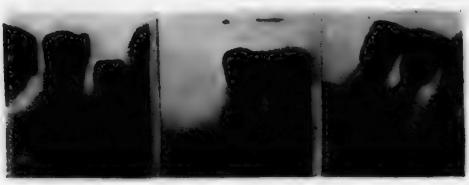

FIG. 24

FIGS. 21, 22, 23 AND 24. A SUCCESS, SHOWING COMPLETE REGENERATION AFTER PERFECT ENCAPSULATION

Fig. 21. The roentgenograms were taken (left) on February 12, 1920, and (right) March 3, 1921. The latter shows complete regeneration. See *fig. 24.*

Fig. 22. The roentgenograms were taken (left) on March 13, 1920 (after six separate electrolytic treatments during a period of a month), and (right) November 23, 1920. Complete regeneration is indicated.

Fig. 23. The roentgenograms were taken (left) on April 23, 1920, and (right) April 30, 1920. The former was taken after the first negative bacteriological test had been obtained; electrolytic medication with iodine was continued. The latter was taken after the second negative bacteriological test had been obtained and the root filled.

Fig. 24. The roentgenograms were taken (left to right) on May 18, and November 11 and 23, 1920. Continuous regeneration is indicated. See *fig. 21.*

Fig. 23. We see the condition on April 23, 1920, when, for the first time, no bacterial growth was obtained. Nevertheless, iodine electrolysis was continued. On April 30, 1920, no growth having developed, zinc electrolysis was used, and the root filled.

Fig. 24 shows the stages of regenerative progress from May to November, 1920. So far as these pictures can be properly reproduced, they tell their own story.

IX. SUMMARY OF GENERAL VIEWS

When pulpless teeth are retained in the jaw, they should be placed in such a condition that they can offer no sustenance to microörganisms that may reach them. To accomplish this purpose (a) all pulp tissue must be removed from the canal; (b) all pathogenic conditions of the periapical region must be eliminated; and (c) the entrance of every foramen must be hermetically encapsulated so that there will be no possibility of ingress to the interior of the root. The encapsulation becomes an insulating defensive armament against possible infection.

38 E. 61st Street

THE BASIS AND ADMINISTRATION OF ADVANCED STANDING IN SCHOOLS OF DENTISTRY

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I. INTRODUCTION

By advanced standing is meant the admission of a student to a school at some point beyond the beginning of the course of study. Some refer to this procedure as a "transfer," but it seems better to restrict the term transfer to those rare cases where students change schools within a school year rather than between two school years. In the latter case the arrangement is primarily between the student and the school he enters. The school he leaves has no part in the matter except to issue credentials, while all the matters of decision must rest with the receiving school. This is based upon the premise, which seems beyond serious discussion, that a student has a full right to leave a school at the end of any year. This is a corollary to the right of a school to dismiss a student at the end of any year. Both of the premises rest upon the generally accepted theory that the tacit contract between a school and a student is an annual one, terminable at the end of any year by either party without prejudice. The old idea of the proprietary schools, that the student belonged to a school, being in a sense a chattel, is now pretty generally discredited.

[1] The author is a special student of professional education and a member of the Dental Educational Council of America.—(W. J. G.)

There are occasionally cogent reasons why a student should change from one school to another. (a) A dental school may not have the entire curriculum in operation. One school on this continent gives only the first two years and sends its students to other schools for the final two years. This plan may be expected to be extended to several other universities in this country, which, due to location in small towns, lack clinical facilities.

(b) A student may desire to change to a school that offers especially good facilities and opportunities in some particular phase of dentistry, to which the student has been attracted subsequent to the time he entered upon the study of dentistry.

(c) A student, by reason of health, may be warranted in changing to a school in a different climate.

(d) Financial reasons may induce a student to change schools, or (e) his family may move to another city and it becomes desirable for him to go with his family.

(f) Occasionally a student attending a school in his home city becomes involved in social relations that take so much of his time and attention that it is advisable for him to change schools.

All of these are real reasons why a student should change schools, but they do not include the usual incentive that leads students to change. In by far the majority of cases the changes are brought about by lack of scholastic success. Rarely this lack of success may be ascribed to personal misunderstandings between students and individual teachers, but the greater number of students who change schools may be included in the expressive euphemism, "lame ducks." Since this is so, we must have constantly in mind, in our discussion of advanced standing, a procedure that will involve a reasonable solution of the "lame-duck" problem.

II. THE BASIS FOR ADMISSION TO ADVANCED STANDING

A correct conception of advanced standing must be based upon certain premises.

(1) When a school grants its degree to a student, it guarantees the qualifications of that student to both the profession and the public, and takes the responsibility for that student's shortcomings just as it profits by his capabilities. To the profession and the

public, the degree of a student who has been in a given school, for the senior year only, is indistinguishable from the degree of a student who has spent the entire four years in that school.

(2) If a school is to maintain that rather elusive criterion known as its "standard," it must be sure that the years spent in another school by a student who seeks advanced standing are fully equal in all respects to the corresponding years of its own curriculum; because, when it graduates this man, it must guarantee his entire training. The situation is comparable to that of a man who endorses another's check.

(3) The school that admits a man to advanced standing must guarantee his preliminary education. Therefore, it must make sure that such a student had completed, before entering upon his first dental year, the preliminary education required of the members of the class he seeks to enter. To *assume* this to be true is unsafe. The fact that he comes from a school that advertises a claim to require a given preliminary education for entrance is not a guarantee that any particular man possessed that education, because administration of entrance requirements is not always beyond criticism. Unless the school he first entered verified his entrance credentials, there is a chance that fraudulent credentials were used. The school that receives a man to advanced standing must, to protect itself, either secure a certified copy of his entrance credentials direct from the preparatory school, or obtain, from the dental school where he first entered, a certified copy of the entrance credentials on file in that school, with the additional certification that they have been verified.

(4) Admission to advanced standing is essentially the same thing as promotion, except that it is promotion from a lower class in another school. Therefore, when a student from another school is admitted into an advanced class on the same basis as students who have been constantly in attendance, the school so admitting a student thereby tacitly certifies that the school from which the student came is in all respects as good as the school in which he is admitted to advanced standing, if such admission is without examination or requirement of additional work. If this principle is thoroughly appreciated by a school that is trying to maintain high standards, it will refuse to admit to advanced standing on credentials from schools it considers to be weaker schools.

(5) A school maintains a curriculum on the presumption that the knowledge and training gained by a student in the earlier years of that curriculum are essential, as a preparation to the work of the later years. If this is not true there can be no reason to require a student to take the earlier years.

(6) A school maintains courses of a given excellence because it believes that courses at least as good as its own are necessary in preparation for its more advanced courses; and it bases its advanced courses upon a minimum of knowledge and training represented by its preceding courses. If this is not true, there is difficulty in justifying the existence of these earlier courses.

(7) A school requires, in each year of its curriculum, certain courses and subjects because it believes a knowledge of these particular subjects is essential in preparation for the subjects of the next succeeding years; otherwise, there is no valid reason for teaching these subjects.

III. ADMISSION TO ADVANCED STANDING IS EQUIVALENT TO PROMOTION. STANDARDS OF PROMOTION

Admission to advanced standing is equivalent to promotion, and our conception of its theory and administration must rest upon the same fundamental ideals as those we exercise in promotion. Unless this doctrine is accepted, a school will lower its standards and ideals whenever it admits a man to advanced standing.

What should be the standard of promotion in any school that is organized and administered on a really educational basis? This is a pertinent inquiry here, since these same standards should also be applied to the applicant for advanced standing. It seems reasonable that, before promoting a student to an advanced class, the administrative officers of the school should be convinced in regard to the following particulars.

First; that the student has had a preliminary education, before entering the dental school, that will enable him not only to be successful in the dental course but also to take a reputable position in the profession.

Second; that he has had instruction and training requisite to the work he is about to undertake, i.e., has taken and passed courses in

all the subjects that are prerequisite to the subjects and courses of the year he is about to enter.

Third; that the courses he has passed have been of sufficient excellence to permit him to profit by the instruction of the succeeding year.

Fourth; that, if he has conditions and failures, the total of these is not beyond his capacity to carry in addition to the regular work of the succeeding year.

Fifth (including all the preceding); that the student has sufficient ability, character, industry, knowledge, and training, to give reasonable promise of being able to pursue successfully all the work of the year to which he is to be admitted, and then to complete the course and finally to become a successful and reputable member of the profession.

IV. CIRCUMSTANCES AFFECTING ADMINISTRATION OF ADVANCED STANDING

How shall a school administer advanced standing to assure, on the side of the school, maintenance of its standards and, on the side of the student, a reasonable probability of success?

Since the school must depend for its deductions upon written credentials of the previous educational record of the student, it is evident that the starting point is certainty of the authenticity of these credentials. Unfortunately, experience has shown that occasionally a dental student is a party to falsification of credentials, either by presentation of credentials to which signatures have been forged or by alteration of details within the body of a credential the signature to which is genuine. Therefore, a credential that has passed through the hands of a student should be accepted only as a memorandum, the details of which must be verified immediately by direct communication with the school and officer issuing the credential.

The *first* step in administration of advanced standing is, therefore, verification of the details of credentials regarding both the preliminary education and the work previously done in dental schools. This requires considerable time; meanwhile, the further steps in administration may be taken, subject to the verification of credentials.

The *second* step is to ascertain whether the applicant has passed courses in all the subjects which have been taken by the members of the class he seeks to enter. If he has not passed all such courses, it is evident that he is less well prepared, as to both knowledge and training, than are the regular members of the class he seeks to enter; and that therefore, at the outset, he is handicapped.

The *third* step is to ascertain whether the courses he has taken and passed in various subjects are reasonably equivalent in extent, content, and excellence, to the corresponding courses already passed by the class he seeks to enter. If those courses are markedly deficient as compared to the courses taken by the regular members of the class, it is clear that he will be at a disadvantage in the class and in competition with his classmates.

The extent of his courses can be approximately judged by the number of hours of instruction; therefore, every credential bearing on advanced standing should carry the number of hours for each course that is certified.

The content and excellence of courses taken in another school are more difficult to determine, but some sources of information are (a) the general reputation of the school from which the applicant comes—helpful, but not alone adequate, since every school has some weak courses; (b) the catalogue description of the course, which cannot be very satisfactory in the present status of dental school catalogues; (c) note-books of the applicant; (d) informal talks with the applicant; but, most important of all, (e) the opinion of your own teacher in the subject under consideration, for every teacher in charge of a subject in any dental school should know the instructor, and be familiar with the course, in his subject in the other dental schools.

The information as to the extent, content, and excellence, of any particular course in any given school, when once thoroughly ascertained, can be used for some years as a basis for consideration of other applicants from that school, on the assumption of a fair degree of stability in the curriculum of any school; although we must remember that both the instructors and content of courses are constantly changing, happily usually for the better.

The *fourth* step is to reach a conclusion as to the ability and industry of the applicant. These two characteristics of a student are so intimately associated that they cannot well be considered separately in this connection. Aside from the statements volunteered by the officers issuing the credentials, the best source of information is the general trend of the grades attained by the student as shown by his credentials. While grades do not serve as a good criterion of either ability or industry alone, they do serve as a fair indication of the combination of the two. But to make an adequate deduction from grades attained in another school, an administrative officer must have all the grades, not simply the final grade. Hence, every credential should show all grades in each subject, i.e., if a student received, in the first instance, a failure or condition in a subject and later made it up, the fact of the failure or condition, and not simply the final grade attained in making up the deficiency, should appear in the credentials. To neglect to state the fact of conditions or failures, later removed, is to deceive the school that receives the student, inasmuch as the full educational record is not given.

A further basis of judgment, and really more critical than mere numerical grades, is a statement of the exact status the student would have if he had remained in the school from which he comes. Therefore, every credential for advanced standing should carry an unequivocal statement of the privileges of the student. Can he go on with his class without or with conditions, or must he repeat a year, or is he denied reëntrance to the school? Until the receiving school knows exactly the privileges of the student in the school from which he comes, there can be no intelligent decisions. A statement that there is "no objection" to his being received is too absurd to be of any value. This verbiage is based on the old chattel theory of the proprietary schools. Certainly no school of standing has any objection to some other school taking one of its "lame ducks." The "honorable dismissal" is just as futile, because it is meaningless from a scholastic standpoint; and usually indicates simply that the student has paid his bills.

The *fifth* step is to determine something of the personality and character of the applicant. Certificates from friends are of very little value, but his character can be judged to some extent by the

disciplinary actions his former school has imposed. Therefore, every credential should carry a statement of all disciplinary actions. His personality can be judged only in an interview, and this is often difficult to obtain, since most advanced-standing cases are carried on by correspondence.

The *sixth* step is to ascertain the reason why the student wishes to change schools.

The foregoing discussion is based on the assumption that the student has a clear record in the school he leaves, and wishes to change schools for a good reason.

V. TREATMENT OF UNREMOVED CONDITIONS

We now come to a consideration of the "lame-duck" group of students, who have unsatisfied failures or conditions in the schools where they have recently been students.

It seems necessary to discuss the treatment of unremoved conditions. The required action regarding unremoved failures is too patent to need any discussion. Some schools are in the habit of giving examinations to remove the conditions of a student who is admitted to advanced standing. Such a procedure seems to be very doubtful in its educational ethics. Obviously the examiner cannot know intimately the content of the course in which the applicant has incurred a condition. He cannot know the character of the teaching on the many controversial points that are inherent to every subject. Hence, his viewpoint of what is a correct answer may be at variance with what has been taught the student and, to this extent, the examination is unfair to the student. The usual outcome is that the examination must be on the subject, not on the course taken. The impression prevails that examinations of this sort are so easy as to become merely perfunctory and therefore practically worthless educationally. In schools that follow this procedure, the almost invariable result is that the student passes the examination; and these results indicate that such examinations are devices for removing technical disabilities rather than for ascertaining the capabilities of the students.

Schools of good standards and ideals will not consent to remove, by examination, conditions incurred in other schools. Such schools

will take the stand that, unless the condition is removed in the school where it was incurred, it must be considered a failure; and that a student either has credit in a subject or a failure. In the latter case, the course must be repeated when he changes schools. Any school that follows the device of removal, by examination, of conditions incurred elsewhere is at once open to the suspicion of maintaining low standards and a desire for quantity rather than quality of students.

A middle ground is an offer from the receiving school to conduct an examination set by the school where the condition was incurred, the examination responses to be returned to the former school for grading. This plan is not at all satisfactory, since such an examination must necessarily be entirely written and cannot include the oral or practical phases that often are highly desirable in order to secure a true index of the student's ability. Moreover, the school from which the student is received might well demur at giving such an examination, since the student, by applying for advanced standing in another school, has, by this act, left his former school; and is therefore no longer entitled to its educational privileges, of which examinations are one.

It is fair to say that nearly all schools of better standards and ideals now refuse to admit a man to advanced standing unless he has a clear record, excepting only courses (in which he lacks credit in the school from which he comes) that, by reason of differences in curriculum, are parts of the regular work of the year the applicant seeks to enter. Also, some schools refuse to admit to advanced standing a student who has barely passed his courses in his former school. Such a record indicates a uniformly weak man and the likelihood of his success is problematical.

We must now consider what amount of "back work," in addition to the regular work of the year about to be entered, is likely to be successfully carried by a student entering advanced standing. This work may be in courses the class he seeks to enter has had, but which the applicant has not had by reason of differences in the curricula of the two schools concerned, or it may be in courses not passed in the former school, or in a combination of both sorts of deficiency.

In the matter of promotion from class to class in professional schools, it seems generally accepted that 20 per cent in extra work is the ex-

treme maximum a student can be expected to carry, in addition to the work of any year of the curriculum. Certainly the man admitted to advanced standing should be put under no greater handicap. It is doubtful if as much as a 20 per cent handicap is justifiable. Every school has its peculiarities in methods. While the regular members of the class have become accustomed to these, the student who is admitted to advanced standing is not conversant with the idiosyncrasies of the school. Therefore, he cannot be expected to work as effectively as those students who have had the advantage of preparation in courses which are more or less closely correlated with the particular courses of the year about to be entered. It seems reasonable to conclude, therefore, that, if 20 per cent in additional work is the extreme handicap for students who have just come through the earlier years of that school, it is too great a load for the man admitted to advanced standing. Hence, probably 10 per cent in additional work is all that any such man is likely to carry successfully. In other words, no man should be admitted to advanced standing in a class, who is obliged to repeat more than 10 per cent of the work of the previous year.

Since admission to advanced standing is, in essence, promotion, a school, out of fairness to its own students, should grant no greater concessions to a man admitted to advanced standing than to one of its own students.

VI. MIGRATION OF "LAME DUCKS" TO "SAFE HARBORS"

The experience of most dental schools shows that the men admitted to advanced standing usually are unsatisfactory students. While it is true that some students change schools for good and cogent reasons, and even occasionally are in search of a better school; yet, for each one of these, there are ten who change schools in the hope of finding easier conditions. A majority of the students who change schools are either poorly prepared, indolent, or lacking in intellectual capability; hence it is no wonder that they are usually found to be unsatisfactory students. If we can get, in the schools, a clearer conception of what advanced standing really is, and a more careful administration in regard to it, we shall be able, by selection, to bring the average quality of advanced-standing students up to the average level of the classes they enter.

There is no phase of professional-school administration that so jeopardizes the standards and reputation of a school as does carelessness in granting admissions to advanced standing. Let us take a few concrete examples to illustrate this statement.

Mr. A in X school has been a weak student for a year or two, or three. Every instructor knows this. He may have a string of failures and conditions unremoved, and it may be pretty well understood that he will either be dropped at the end of the year or required to repeat a year. He changes to Y school and goes right along without losing time. The officers and instructors in X school immediately form the opinion that "Y school takes our flunkers."

Again, Z school is one which is pretty generally known to be of low grade. Possibly classifying agencies have designated it as a low B, or even a C, school. Y school accepts into its second, third, or even fourth year, students who have completed one, two or three years, respectively, in Z school. Immediately the officers, instructors, and students of Z school claim, with considerable justification, that Z school is just as good as Y school, since Y school admits to advanced standing the students from Z school. They go even further and suggest, to prospective students of Y school, that, instead of going directly to Y school, they enter Z school (which is easier) for a part of their course; and then change to Y school, if they desire to get their degree from a school of higher rating. Such a suggestion is pertinent and convincing when concrete cases can be cited where this procedure has been accomplished.

Mr. B. in X school is a poor student with conditions and failures, and on the eve of being dropped, or possibly already has been dropped. Mr. B. is a fraternity man; and at the beginning of the next year the members of his chapter hear, from him, that he is in Y school, that he removed all his conditions by "snap" examinations, and is now going along with the class to which he would have been denied entrance in the school where he was formerly a student. Not only does the fraternity chapter in X school learn of this, but the word is passed on to the chapters in other schools, that, if any fraternity brother gets into scholastic difficulties, Y school is a haven to which he may turn.

The authorities of Y school, in succeeding years, get applications for advanced standing from widely separated parts of the country and pride themselves that their school is becoming famous; when the simple fact is that the word has been passed along, among the students of various schools, that Y school is a "*safe harbor for lame ducks!*" Inquiry will show that students in any dental school know pretty accurately the location of these "harbors," probably even better than do the instructors. Further, this reputation of Y school is remembered by the students of X school and other schools, after they have graduated and are in the profession; and, for years, they will look on a graduate of Y school as inferior because he came from a "harbor for lame ducks" and may even be one himself. Thus, loose administration of advanced standing in any school diminishes the professional reputation of every one of its alumni; and, even if the officers of a school are indifferent in this matter, it is to the interest of thealumni to force better standards in matters of advanced standing in their schools.

If dental educators can come to realize the situation, and if, through such realization, most or all of these "harbors" can be closed, dental education will be improved in every school; because the students in any school will put in greater effort if they know that, in case of failure in that school, it is very difficult or almost impossible to enter any other school. It is not uncommon in the school with which I am connected for a student, upon being warned of poor work, to say: "If I get thrown out here, I can go to —— or ——," naming two schools which have accepted large numbers of our discards in recent years.

VII. SUMMARY OF SALIENT POINTS

What are the salient points in administration of advanced standing? Succinctly stated they are the following:

(1) Verify all credentials.

(2) Ascertain whether the preliminary education is equal, in every respect, to that required of the members of the class the applicant seeks to enter, i.e., do not admit to advanced standing a man who would not have been accepted if he had sought to enter your freshman class rather than that of the school from which he now comes.

(3) Ascertain whether the applicant has had courses in all the subjects which have already been taken by the class he seeks to enter, i.e., do not admit students to advanced standing, year for year, without knowing that the years are fully equivalent to your own.

(4) Satisfy yourself that the courses taken are reasonably equivalent in extent, content, and excellence, to the corresponding courses in your own school.

(5) Ascertain under exactly what conditions the applicant could return to the school where he has been a student, and do not accept "no objection" and "honorable dismissal" as commendatory of an applicant's desirability as a student.

(6) Refuse to give examinations to remove conditions incurred in another school.

(7) Refuse to give the applicant any more favorable terms in regard to "back courses" than you would give your own students.

(8) Refuse to give the applicant more favorable terms than would have been given him by the school from which he comes, i.e., if he would have had to repeat certain subjects or a year in the school from which he comes, do not require less of him in your school. This is necessary to protect your reputation as to standards.

(9) If the record of the applicant shows many conditions and failures, although later removed, consider very carefully whether his record indicates a standard of ability and industry commensurate with that which you exact of your own students. If it does not, then refuse him. In a word, do not dilute the quality of your own student body by taking in an unknown who is evidently of low capability.

(10) Do not accept students who have been expelled from some other school for dishonesty, either economic or scholastic, or for moral delinquency. To do so is an offense to every member of your own student body, and one may properly infer that *you* consider your own student body of lower moral grade than that of the school from which the accepted student was expelled.

(11) Only in extremely rare cases admit any one to your senior class. A student who has been three years in one school and but one year in another school is more a product of the former than of the latter school, although he holds the degree of the latter school. He

cannot be expected to have the ideals, spirit, or loyalty, of your school nor to be a fair example of your product.

(12) Finally, admit students to advanced standing only with a view to maintaining the ideals and standards of your school, not with a view simply to increasing your numbers or income; for the future of dentistry, and the reputation of dental schools and their graduates, is going to depend upon the *excellence* of the graduates, not upon their *number*.

THE INCIDENCE OF DENTAL CARIES IN THE PRE-SCHOOL AGE

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I. INTRODUCTION

Numerous studies have determined the incidence of dental caries in various populations and in different age groups. Brief reference to one that is representative may not be amiss. Kent states that "dental caries is the most prevalent of all human diseases, being found in a progressing stage in the mouths of at least 95 per cent of our school children."[1] A search has failed to reveal a record of a study relating to children *under* school age. If any such has been published, it is not of general value since it is not readily accessible.

A study of dental caries during the pre-school period should be of more than passing interest. It is the time of the mass assault of caries upon the deciduous teeth. Any study of caries at this age requires a body of established data, as normal, for comparisons and contrasts.

II. PROCEDURE

This paper, on the incidence of dental caries in children under school age, presents data without reference to the etiology or other relationships of caries. It is a by-product, however, of a study upon caries in association with the dietaries of the children examined. (The results of the main study will be reported in another paper, which

[1] Kent, Edwin N. (Supervisor of Mouth Hygiene, Massachusetts Department of Public Health): *The Commonwealth*, September–October, 1920; Massachusetts Department of Public Health, Boston.

will detail the procedures and conclusions derived; see the next succeeding issue of this JOURNAL.) Here it need be stated merely that the teeth of the children were examined in good daylight *by medical men of good training*, each of whom had been interested in the study. They showed enthusiasm, and coöperated effectively. The teeth were bared with a wooden tongue-blade and each tooth was separately examined for the presence of caries. Any caries was reported (by dictation to a secretary) for the particular tooth involved. In many cases the writer checked the findings, often probing with a sharpened splinter of wood. It was uncommon to find incorrect reports.

This method of examination is not above objection, of course. If routine dental instrumentation had been employed, and if the observations had been made by trained dentists, a greater number of cases would assuredly have been found, especially in very early cases where the degree of enamel destruction was still quite small and could be determined only by careful dental probing. However, in a careful collateral study with the Children's Department at the Deaner Institute, only 8 per cent of the number of cases were missed, and less than 0.5 per cent of the number of teeth were wrongly diagnosed. Assurance has been given by a clinician in children's dentistry that, if caries is so slight in degree, in a deciduous tooth, that only expert examination can discover it, the decay will be obvious to direct inspection, without instrumentation, within two or three months.[2] This means that only the cases seen very early in the decay process were apt to be overlooked; and, since caries remains obscure for so short a time, it is obvious that only a small percentage of cases of initial caries would be missed in the procedure followed.

It is understood, then, that this paper relates to a study of dental caries that was sufficiently advanced in each case to be obvious to the untrained eye by direct inspection alone.

The school districts covered by the report are situated in all parts of Kansas City and represent an excellent mixture of all the grades of human enterprise in child rearing, and of all classes of social distinction. Children in the white population alone were examined in this study. The data have been assembled independently for each school district in which the examinations were made, and a cumulative

[2] Personal communication from Dr. T. B. McCrum, Kansas City, Mo.

report compiled covering nineteen districts and the 1,197 cases studied.

The data taken from the record cards, for tabulation, are the age of the child; the name of the school; the number of teeth erupted; the presence or absence of caries; and the number of carious teeth,

Data pertaining to incidence of dental caries (a cumulative report)

	AGES BY YEARS							
	0–1	1–2	2–3	3–4	4–5	5–6	3 to 6	0 to 6
(1) Mouths with teeth	80	218	223	220	224	232	676	1,197
(2) Total number of erupted teeth	301	2,386	4,093	4,388	4,468	4,742	13,598	20,378
(3) Average number of erupted teeth per mouth $\frac{(2)}{(1)}$*	3.8	11	18	20	20	20.4	20	17
(4) Number of mouths found to be carious	0	5	23	67	85	145	297	325
(5) Percentage of the total number of mouths found to be carious $\frac{(4)}{(1)}$	0	2.3	10.3	30.4	38	62.5	44	27.2
(6) Total number of teeth found to be carious	0	10	62	249	343	622	1,214	1,286
(7) Percentage number of carious teeth, in the total number of erupted teeth $\frac{(6)}{(2)}$	0	4.2	1.5	5.7	7.7	13.1	8.94	6.31
(8) Average number of cavities per mouth, in the total number of mouths $\frac{(6)}{(1)}$	0	0.05	0.27	1.13	1.53	2.68	1.80	1.07
(9) Average number of cavities per mouth in the total number of carious mouths $\frac{(6)}{(4)}$	0	2	2.7	3.72	4.04	4.29	4.09	3.95

* The numerals in fraction arrangement, in this column, represent the lines of values in this table from which the data for the respective calculations are taken.

if any. The findings were separately recorded for each year, from zero to six. Two columns of totals were arranged: the first covered a period that might be called the age of the mass incidence of deciduous dental caries, comprising the years from three to six; the other, covered the entire pre-school age from birth to six years. The last yearly

period, from five to six years, registers the cumulative results of influences that prevailed prior to school age.

The data in the accompanying table, covering the 1,197 cases, tell their own story. Attention may be called, however, to a few of the more significant findings (a–f).

III. DISCUSSION OF THE FINDINGS

(a) More than a quarter (27.2 per cent) of the number of these children presented dental caries, averaging over one tooth decayed (1.07) for each mouth examined, and almost four decayed teeth (3.95) for each of the carious mouths. Of all the teeth erupted, 6.3 per cent presented obvious caries.

(b) In the last three-year period (3–6) before the children enter school, i.e., in the "caries period," 297 mouths were carious out of the 676 examined. This is 44 per cent, or nearly one-half. Over twelve hundred (1,214), or one-twelfth (8.94 per cent) of the total number of erupted teeth, were carious, the carious mouths averaging a trifle over four decayed teeth each (4.09).

(c) Even during the one-two year period, there were five carious mouths that averaged two diseased teeth each.

(d) In the next year, two-three year period, there was a sudden appearance of caries in more than one-tenth (10.3 per cent) of the number of children. Even at this age the carious mouths averaged nearly three diseased teeth (2.7) each.

(e) But it is in the three-four year period that the mass appearance of the disease is forced upon one's attention. Sixty-seven, or 30.4 per cent, of the 220 children of this age had dental caries. They averaged over three and one-half involved teeth (3.7) in these cases.

(f) During the next year the incidence index is higher; and in the five-six year period it presents pronounced proportions. This five-six year period, which definitely represents the dental condition of the children at the close of the pre-school age, should receive particular study because it presents the obvious results of influences acting up to that time. Furthermore, the youngsters are now about ready to begin their schooling, and the findings in this year represent definitely the condition they will present upon entering school. Until then, loss of time has little or no importance in their lives. Once in school,

absence from studies is a handicap to the pupil and an economic loss to the community. On the basis alone of loss from trivialities associated with dental caries—such as toothache, abscess, or time required for receiving dental attention—the problem is an important one. Considered, however, in the light of consequences of major handicaps that can and do result from dental disease—rheumatism (often called "growing pains"), heart disease, and mal-development—the problem is distinctly serious. There have been reports[3] of improved mental alertness from the treatment of the tooth troubles of school children, and a consequent reduction in the number required to take grades a second time. Dental disease evidently plays a serious rôle in the lives of the little school children, and the economic consequences of dental disease in children are obviously important.

The five-six year period presented 232 children for study. They averaged a trifle over twenty erupted teeth, many of which were erupted permanent molars. One hundred and forty-five of the children, or nearly two-thirds of the number (62.5 per cent) had caries. They averaged over four (4.29) decayed teeth per carious mouth. More than one-eighth (13.1 per cent) of the number of erupted teeth were carious. For all the mouths of this period there was an average incidence of nearly three (2.68) decayed teeth per mouth.

To prepare these youngsters dentally for their school life, almost two-thirds of them would have to be treated. In the average child needing treatment, more than four of his teeth would require important surgical attention. In a city of 325,000 population (Kansas City, Missouri, for example), more than 8,000 children would enter the first grade each year.[4] Over 5,000 of these children would need dental attention, and over 21,000 teeth would require surgical interference. This is indeed an economic problem as well as one of humanity.

926 Taylor Street,
San Francisco, California.

[3] Ebersole, Wm. G.: The human mouth, and its relation to the health, strength, and beauty of the nation; *Dental Cosmos*, 1911, liii, pp. 787–803. O'Neill, Cordelia L.: Oral hygiene as it appeals to educators; *Dental Cosmos*, 1911, liii, pp. 1393–1399. Fones, A. C.: Report of five years of mouth hygiene in the public schools of Bridgeport, Connecticut; *Dental Cosmos*, 1919, lxi, pp. 607–618.

[4] Statistical and Research Department, Board of Education, Kansas City, Missouri. First grade enrollments were as follows: 1918–'19, 8313; 1919–'20, 8421; 1920–'21, 8146.

THE INFLUENCE OF ALKALINE AND ACID TOOTH PASTES UPON THE ACTIVITY OF SALIVARY ENZYME

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I. PROCEDURE

Tests were made with both kinds of tooth paste in dilutions equaling those obtained in brushing the teeth, namely, one to twenty. The paste was freely mixed with distilled water in a closed flask. The mixture was allowed to stand about an hour, and then filtered, exposure to room air being avoided.

Both kinds of filtrate were subjected to the following tests, and the acid filtrate to additional tests to be described subsequently:

(1) Reaction (a) to litmus and (b) congo red. (c) H-ion concentration.

(2) Reducing properties.

(3) Reducing properties when mixed with sugar-free starch suspension (1 per cent).

(4) Reducing power in the presence of saliva and sugar-free starch suspension.

In all the reduction tests, filtrate was thoroughly mixed with an equal volume of sugar-free starch suspension, warmed at 37°C. for fifteen minutes, and then tested by Fehling's test for the presence of reducing substance. All Fehling tests were checked with the newer Benedict method.

II. RESULTS OF EXPERIMENTATION WITH ALKALINE FILTRATE (1:20)

Negative results for reduction were obtained with alkaline paste and sugar-free starch suspension, and also with the alkaline filtrate alone, indicating that by itself the latter does not possess reducing properties.

Aside, of course, from its being alkaline to red litmus, the reaction of the filtrate to congo red was negative; its H-ion concentration was pH 8.2. In preparing filtrates for H-ion determinations, it is necessary to use freshly distilled water and to avoid contact with room air. Distilled water tends to pick up carbon dioxide from the air and, upon standing, even when stoppered, its reaction changes.

Alkaline filtrate, mixed with human saliva and warmed with sugar-free starch suspension, showed, when tested with Benedict's and Fehling's solutions, a positive reduction. This positive test showed that the alkaline paste did not interfere with the digestion of starch by salivary amylase.

III. RESULTS OF EXPERIMENTATION WITH ACID FILTRATE (1:20)

Reaction: acid to blue litmus; H-ion concentration, pH 5.3. When warmed at 37°C., and tested with sugar-free starch suspension, a positive result was obtained in the reduction test. This positive reduction is interesting and requires further consideration. On first thought it seemed that the reducing substance was due to some ingredient in the acid paste, because glycerol, pyrocatechin, and closely related substances, which are used as mild antiseptics, sometimes give a positive reaction with Fehling's solution. On going into the matter more deeply, however, the real reason for this reduction became apparent. Saliva contains a considerable amount of mucin. Mucin is a glycoprotein. Glycoproteins are readily hydrolyzed by acids into simpler proteins and into such reducing substances as glucosamin.[1]

The acidity of the acid paste used in this research is due to acid potassium tartrate. The percentage of acid potassium tartrate was determined in weighed samples by titration against sodium hydroxide, using phenolphthalein as an indicator. In a dilution of one to twenty,

[1] These well-known facts are given in detail in any good text-book of physiological chemistry.

this percentage was 0.077. A solution of acid potassium tartrate of this strength was next added to an equal volume of sugar-free starch suspension, and the mixture warmed at 37°C. for fifteen minutes, and then tested in the usual manner. The result here also was positive, showing that this acid salt is capable of cracking the starch molecule and producing a reducing cleavage product.

Tests were made with acid filtrate against dog saliva, which is known to be free from amylase. The animal's saliva was readily obtained under pilocarpine medication. This in turn, when warmed in the usual manner with acid filtrate and tested with Benedict's and Fehling's solutions, resulted in typical reductions.

Lastly, dog saliva, plus an equal volume of 0.077 per cent solution of acid potassium tartrate, was subjected to the same procedure, resulting also in reductions.

These facts clearly show that the positive reduction tests with saliva, reported above, were due to "activity" of the acid in the paste and not to that of salivary amylase; furthermore, the H-ion concentration of the filtrate (pH 5.3) is unfavorable for the activity of salivary amylase. Mathews[2] has shown that acidity above pH 6.7 inhibits the activity of salivary amylase and that all activity ceases at pH 4.

On the other hand, the alkaline filtrate did not interrupt salivary digestion. A similar conclusion has been stated by Prinz.[3]

IV. CONCLUSIONS

The alkaline paste used in these tests did not inhibit the action of salivary amylase.

The acid paste used in these tests inhibited the action of salivary amylase.

The positive reduction tests obtained in mixtures of the acid paste, starch, and (dog) saliva, were due to the production of reducing substance from the starch and from the salivary mucin by the hydrolytic action of the acid in the paste, not by the action of salivary amylase.

[2] Mathews: Physiological Chemistry, p. 331 (1915). Wm. Wood and Co., New York.

[3] Prinz: Dental Materia Medica and Therapeutics: 5th Edition, p. 287 (1917). C. V. Mosby Co., St. Louis, Mo.

SCIENTIFIC PROCEEDINGS OF DENTAL AND STOMATOLOGICAL SOCIETIES

THIRD SECTION

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A LIST OF THE NAMES OF THE ORGANIZATIONS THAT HAVE VOTED TO MAKE THE JOURNAL OF DENTAL RESEARCH THE MEDIUM FOR THE OFFICIAL PUBLICATION OF THEIR SCIENTIFIC PROCEEDINGS

Association of the Allied Dental Societies, Inc.

American Academy of Dental Science

Massachusetts Dental Society

Harvard Odontological Society

Boston and Tufts Dental Alumni Association

First District Dental Society of the State of New York

Metropolitan District of the Massachusetts Dental Society

Harriet Newell Lowell Society for Dental Research, of the Harvard University Dental School

San Francisco Society for Dental Research

Colorado Society of Periodontists

American Academy of Periodontology

New England Society for the Promotion of Dental Research

American Society of Oral Surgeons and Exodontists

William Jarvie Society for Dental Research, of the School of Dentistry of Columbia University

Columbia University Biochemical Association

In the case of the last named organization, only the dental and stomatological parts of its proceedings will be published in this Journal.

A standing general invitation is extended to dental and stomatological societies, everywhere, to add their names to the above register.

COMMON TUMORS THAT OCCUR ABOUT THE MOUTH[1]

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I. DEFINITIONS; CAUSES; GENERAL TYPES

Before we proceed to a discussion of the commoner tumors that occur about the mouth, it would be well to consider the question, what is a tumor, particularly as many of you have not yet had a course in pathology. A tumor is a local overgrowth of tissue that fulfills no useful purpose. You might think that this definition applies to a boil, or to a swollen cheek following dental abscess; but that is not so, for both boil and swollen cheek are evidences of local combats between invading bacteria and defending leucocytes. A tumor represents no such conflict, it merely grows and does nothing useful.

What causes these growths? We do not know. We can guess along a variety of lines, but the fact remains, we do not know. We do know, however, that certain body cells may suddenly begin to multiply and form local overgrowths of certain types of tissue. These cells may belong to one of three classes: totipotential, multipotential, and unipotential. The ovum and its immediate derivatives are cells of the first class; they may develop into any type of body-cell. The cells of the three primitive germ-layers belong to the second class; they have a multiple potentiality and are capable of forming a variety of different cells, but they can not form *any* variety—they are limited to the restrictions of the particular germ-layer to which they belong. The unipotential cells, of the third class,

[1] Informal lecture at a meeting of the Harriet Newell Lowell Society for Dental Research, Harvard Dental School, Boston, Massachusetts, April 13, 1922.

are the commonest type, because most adult cells fall under this category. They are capable of producing but one type of cell, a type resembling themselves. That is to say, unipotential epithelial cells of the liver parenchyma produce only liver parenchyma; those of the retina produce only retinal cells; and so on.

Now let us suppose that something stimulates one of these cells to start multiplying—to form a little colony of similar cells that are quite useless to the body that harbors them. If that cell be totipotential, it may form a very complicated and highly organized colony, even to the extent of producing a very close approximation to another individual. Such a tumor would be an *embryoma* or a *teratoma* according to the degree of differentiation, the former being but slightly, the latter highly, differentiated. A multipotential cell forms a less complex tumor, but one composed of several types of cells, which is called a "*mixed tumor*," and is most commonly found about the salivary glands and the kidney. If a unipotential cell be the parent of the tumor, all its cells will be of the same type, forming a *fibroblastoma*, an *osteoblastoma*, a *lymphoblastoma*, etc.

We do not know what causes these tumors. Some of them are present at birth, some occur in childhood, and others do not appear until the latter half of the patient's life. The last type is often of the type known as "malignant;" apt to kill by spreading to other parts of the body, and characterized by rapid growth, by this tendency to spread, or "metastasize," and by the embryonal type of its component cells. Tumors may be brought about by embryonal cells lying dormant for a time and then starting to grow, in response to some stimulus like constant irritation from smoking, or nursing a child, etc. Or, cells or groups of cells may be included between two fusing masses of tissue in the course of embryonal life and later begin to grow. Tumors have been experimentally caused by repeatedly injecting irritating chemicals beneath the skin of a rabbit's ear. They may also represent an attempt on the part of a group of senescent cells to renew their youth by growing rapidly. No microörganism has ever been found to cause tumor growth.

II. THE IMPORTANCE TO THE PATIENT OF RECOGNITION OF ORAL TUMORS BY THE DENTIST

The importance of tumors to the dentist may not be great from the active or curative standpoint; but he may be instrumental in discovering their presence, where they have been unsuspected by the patient, and in putting that patient on his guard. Oral surgeons may be called upon to operate upon some of these tumors; the prosthetic expert may have to

repair the deficiencies left in the oral cavity by such operations, by supplying bridges, dentures, etc. If you recognized a cavernous *hemangioma* as such, you would know enough to avoid wounding it and causing serious or perhaps fatal hemorrhage. If you recognized an *epulis*, you would know how to deal with it and what the chances were for recovery after its removal. If you knew a *carcinoma* by sight, you would be able to impress its importance upon the patient and to refer him at once to one of your medical brethren for treatment.

III. THE IMPORTANCE OF ORAL EMBRYOLOGY FOR AN UNDERSTANDING OF THE NATURE OF ORAL TUMORS

In order to understand tumors of the mouth, you must remember that the embryology of that cavity is of great importance. You will recall that it is a veritable battle-ground of germ layers, as they become pocketed or grow out into fusing arches. During this process of pocketing and fusing, various accidents may happen, which may cause a displacement of tooth germs, or include some cells meant for the production of skin or hair, let us say, in a place where skin or hair have no use, and may be most inconvenient. In such a way tumors come to grow about the mouth with comparative frequency, usually a type belonging to the class of teratoma, or teratoid growths.

As to the embryology of the teeth: you will remember that they are formed in a mould of down-growing ectoderm, called the enamel organ. This has an outer layer of cuboidal cells; an inner layer of cylindrical cells, the "ameloblasts," which form the enamel; and an intermediate layer of branching, mesenchymoid, pulp-cells. This organ becomes hollowed-out by a mesenchymal plug of out-growing tissue, which indents its lower surface and gradually pushes into it. The outer layer of cells of this future dental pulp becomes differentiated into columnar cells, the "odontoblasts," which form the dentin.

IV. TYPES OF COMMON ORAL TUMORS

Supposing a group of cells of the enamel organ become lawless and, instead of forming a tooth, form a sac, or a series of sacs. Such hollow cysts will form a tumor, with possibly considerable substance. The ameloblasts may form enamel; the odontoblasts, dentin; but no true tooth will result, merely a jumble of tooth-like elements. This form of growth is called a "*dentigerous cyst*," a form of tumor which may occupy a quarter or more of the alveolar process, and form a weak spot in the jaw. It may

contain abortive teeth, masses of epithelial cells, fluid, dentin or enamel. Or, we may have an enamel organ growing in a lawless fashion, forming groups of rudimentary enamel organs, and ultimately causing a spherical tumor that may project into the mouth. It may or may not contain enamel, and is called an "*adamantinoma*," or "*adamantoblastoma*." It is essentially harmless, but may be very inconvenient. Here the embryonal tissue has persisted as such, and groups of enamel organs are present where they long since should have disappeared in the course of development. Or, as a third alternative, such persisting enamel organs may stimulate the growth of odontoblasts, which produce masses of dentin, groups of fused toothlike structures, with a pyramidal mass of fused roots at their base. Such formations are known as "*odontomata*," or "*odontoblastomata*."

Perhaps the commonest group of tumors in the oral cavity is that known as "*epulis*," from the Greek words meaning "upon the gum." It is a term that should be discontinued, because it includes several types of tumor. In the first place an "epulis" may be, in reality, a *granuloma*—an inflammatory growth and not a true tumor at all. It projects into the mouth from near the root of a tooth, which it may loosen, and interferes with chewing and eating. It is merely a little fleshy mass, which is apt to be pedunculated, or carried upon a stalk. Microscopically it is composed of granulation tissue, in which one finds many "foreign-body giant-cells," made up of fused endothelial phagocytes, which are attracted by particles of tartar or other foreign material.

Closely resembling this growth is the *fibrosarcoma* of the jaw, or "*giant-cell sarcoma*." This differs from the granuloma in that it is a true tumor, composed of rapidly growing fibrous tissue of an embryonal type, in which are embedded the type of "giant-cells" already described in the case of granuloma. Such tumors may invade the alveolar bone and weaken it, but they seldom metastasize and are considered practically benign, despite the fact that they belong to the malignant group of "sarcomata." The epithelial form of epulis is rather rare and is made up of a local overgrowth of the gingival epithelium, corresponding to a wart on the skin.

Next there are tumors of the epithelium, one form of which we have just mentioned. There are local thickenings of the tongue and lips, which form raised, white plaques, known as "*leukoplakia*." It is generally conceded that they are inflammatory in origin. The important epithelial tumors of the mouth are the "*cancers*," or malignant growths. These are of two types of growth: one, the "*epidermoid carcinoma*," is made up of squamous cells of the mucosa or skin; the other, the "*hair-matrix*," or

"*basal cell*" *carcinoma*, is composed of cuboidal cells from the hair matrices or from the basal epithelium of the skin near the mouth. Both types start as local, grayish, thickened patches, which grow rapidly and soon ulcerate and break down. They are stony hard, and bind the surface layers to the deeper structures, so that one can not move the growth over the underlying parts.

An epidermoid carcinoma may arise from irritation, as from a clay pipe stem (that is constantly hot), or from a broken tooth, or rough crown, or filling. It occurs on the lips, gums, tongue, or tonsillar epithelium, and has the fatal habit of metastasizing to other parts of the body if not promptly recognized and removed. It is usually typified by the presence of greatly overgrown surface layers of epithelium, which tend to form horny whorls of cells, known as "epithelial pearls," and by the fact that the basal layer of cells no longer forms an unbroken layer, but is interrupted at frequent intervals by erruptive down-growths, or plugs of rapidly multiplying cells, which invade the submucous or subcutaneous tissue and spread out in it like a fan. As soon as these tumors invade nervous tissue they become intensely painful.

The "hair-matrix carcinoma" is characterized by its slower, more expansile growth, which may destroy a great deal of tissue, the entire lower jaw, for instance. Unlike its relative, the epidermoid cancer, it does not tend to metastasize, as a rule. If not removed at an early date, such tumors may do a tremendous amount of local damage. As can be seen, they always begin outside of the mouth, where there is hair. Microscopically they are recognized by their similarity to hair follicles, minus the hair root and shaft.

Adenomata, or epithelial tumors of the glands about the mouth, are sometimes seen. They may arise from glandular remains beneath the mucosa, left over from fetal life, and form what are known as "*polyps*." They may form rudimentary *thyroid tumors* at the base of the tongue, near the "foramen caecum," the remnant of the thyreo-glossal duct. Or, tumors may arise from elements of the salivary glands, growths usually composed of "multipotential cells" and hence mixed in type. They resemble pieces of sweet-bread in the gross; microscopically they are found to be made up of glandular tissue, which may or may not exhibit malignant characteristics; of muscle and of cartilage; or of cartilage-like material. They always contain a good deal of fibrous connective tissue.

Tumors that concern the dentist quite materially are those composed of blood-vessels—the *hemangiomata*. There are also *lymphangiomata*, which usually occur congenitally; but we shall leave them out of this

discussion. The hemangiomata may be simple coiled vessels or much dilated vessels coiled upon themselves. In the latter case they are called "cavernous." They may be interposed in the arterial, the venous, or the capillary circulation. It is readily seen that a cavernous arterial angioma would be a source of very dangerous hemorrhage if wounded by a slipping burr. If arterial, they pulsate with the beating of the heart. Hemangiomata are usually deep red to purplish-red, and are quite easily recognized.

Other tumors, particularly *connective-tissue tumors* of the fibrous, fatty, bony, or lymphoid tissues, may occur about the mouth, but we shall not take up the remaining time with their consideration. They are not to be classified as "common." The malignant tumors of the bone and lymphoid tissue may prove to be very formidable, when present, but their bearing upon dentistry is less direct than that of the others we have discussed. The *osteosarcoma*, or malignant bony tumor, starting in the cancellous tissue of the bones of the face or jaw, is a very different affair from the fibrosarcoma we spoke of under "epulis." This tumor is very destructive and tends to metastasize widely.

Various teratomata may occur about the mouth, but they are usually present at birth and are important to us only as typifying what a "toti-potential cell" may do, if it has an opportunity to go wrong. Such tumors may develop to a surprising degree, giving rise to small bodies, or arms, or legs, or both, which protrude from the patient's mouth in a mass that is known as "*epignathus*." They are obviously of little importance to the dentist. If they are small and spherical, however, they may be discovered by you and reported for treatment.

V. THE IMPORTANCE TO THE PATIENT OF EARLY RECOGNITION OF ORAL TUMORS; COÖPERATION BETWEEN DENTIST AND PHYSICIAN

In closing, I should like again to emphasize the importance of your recognizing tumors as such and having them seen by medical men; you may thus win the everlasting gratitude of a patient by discovering a hitherto unsuspected tumor which might ultimately cause death, if not promptly removed; *so keep your eyes open!* Once a carcinoma has penetrated to the lymphatics, your patient has a very slender chance of surviving, even if the primary growth be removed, for it will spread to any or every part of the body; liver, spleen, bones, etc., and ultimately cause death after a long siege of possibly intense suffering. Carcinoma of the tongue, for instance, is notoriously a "bad-actor," and must be treated thoroughly and promptly if you wish to save your patient's life.

ANNUAL ADDRESS OF THE PRESIDENT OF THE AMERICAN SOCIETY OF ORAL SURGEONS AND EXODONTISTS[1]

PAST, PRESENT, FUTURE

FRANK L. WARREN

Los Angeles, California

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I. PAST

Something over four years ago, Dr. M. R. Howard, of Denver, Colorado, conceived the idea that there should be formed, in this country, an organization of the members of the National Dental Association who were specializing exclusively in mouth surgery. Acting upon his inspiration, Dr. Howard, after considerable correspondence, took the initiative in this matter and called a meeting of men he knew were devoting all their time to this special line of work.

This meeting was held in Chicago, on August 5, 1918, during the period of an annual meeting there of the National Dental Association, and was preliminary in character. Dr. Howard was elected chairman pro. tem.; and committees were appointed on organization, to draft a constitution and by-laws, etc., and to report at a dinner to be given the following evening. Pursuant to this plan, a permanent organization of the American Society of Exodontists was effected and a tentative report on constitution and by-laws adopted. A. Bromley Allen was elected president; J. Henehan, vice-president; M. R. Howard, president-elect; C. F. Stowell, secretary; and J. A. Blue, treasurer. The Society then adjourned to meet the following year, to perfect the organization.

The second annual meeting was held in New Orleans, October 20, 1919, during the period of an annual meeting there of the National Dental Association. At this meeting the constitution and by-laws were revised. A committee, consisting of Drs. Howard, Molt, and Warren, was appointed to confer with the trustees of the National Dental Association, asking for recognition and affiliation with that body. This request was granted and

[1] Read at the annual meeting in Milwaukee, Wisconsin, August 11, 1921.

the Society became an official section of the National Dental Association. Dr. M. R. Howard, president-elect, automatically became president at the second meeting. Dr. Fred Molt was elected secretary; Dr. Blue, treasurer; and Dr. Warren, president-elect. The officers were instructed to prepare a program for the next annual meeting to be held in Boston in 1920.

Those who were fortunate enough to be present at the Boston meeting know what a wonderful success it was, particularly since it was the occasion of the presentation of our first program. The papers were excellent and very well received. The surgical clinics, given by Boston men, were all that could be desired.

I wish to give to Dr. Howard full credit for the organization of this Society. Other men may have thought of such an organization or perhaps suggested it, but it is to Dr. Howard we are indebted, for it was he who "had the courage of his convictions," and who spent much time and energy in efforts to organize and perfect what is today one of the strongest and best component parts of the National Dental Association. *All praise to Dr. Howard.*

At the conclusion of our meeting in Boston last year, it was suggested to your president, by Dr. Boyd Gardner, that it might be agreeable to the members of the Society to meet informally two days preceding the regular meeting, at the Mayo Clinic, Rochester, Minn., the Clinic to provide a program. Dr. Gardner's invitation was presented to the members during our banquet, when it was unanimously voted to accept the invitation. Those who attended this splendid meeting know what a wonderful success it was. The program, both scientific and social, could not have been surpassed and all profited greatly from it. In behalf of the Society, I wish to thank the members of the staff of the Mayo Clinic, and Dr. Gardner in particular, for that most excellent program.

II. PRESENT

It was with some misgivings that the officers of the Society decided to add another day to our meeting this year, feeling that perhaps we could not fill a three-day program; but we are now confronted with a program that will occupy every available minute of the allotted time, and we may be compelled to hold some night sessions. Dr. Arthur E. Smith, who arranged our program, is to be congratulated, for, through his broad acquaintance and popularity throughout the country, he has "assembled the very best talent." We are particularly fortunate in having a member who is so well qualified and willing to give much of his time and energy to this very important matter.

As president of this Society, I wish to express my most sincere gratitude to the officers and committees with whom it has been my pleasure to work during the past year. It has never been my good fortune before to work with a group of men who have responded so willingly and who have shown such marked enthusiasm as have these men. I am personally indebted to Dr. Fred Molt, who consented to accept the secretaryship for another year as a personal favor to me, after he had served through one of the hardest years in the Society's history, namely, that of the year of permanent organization (1920).

III. FUTURE

No section of the National Dental Association has more important work ahead for its future than has this, dealing as it does exclusively with "mouth surgery." During the last few years this special line of dentistry has passed through a wonderful process of evolution. We well remember that it has been but a few short years since all that was expected of any specialist in this line of work was the simple removal of teeth. Today it is far different. It is demanded of us that we be equipped and able to perform surgically any and all operations of the mandible and maxilla; surgically remove all infections; and surgically prepare the mouth for immediate denture insertions. We are called upon to remove cysts and tumors, as well as teeth, both simple and complicated. We are also looked upon as "one-hundred-per-cent" diagnosticians, as well as experts in the art of radiographic interpretation. The fact is that, today, the demands upon the members of this specialty are far in advance of the teachings in the dental schools of this country. Therefore, as a result, what is needed is advanced post-graduate schools with a standard form of teaching the art of modern mouth surgery. These schools should be properly organized, systematized and standardized, so that when a dentist completes such a course, he would have a broad understanding of all phases of the work, and of the most modern technic and methods of procedure as taught by our leading authorities. If such schools were formed, they should have the endorsement and hearty support of this organization.

This Society, as a result of the process of evolution, has outgrown its present name; one that is much broader, and would more fully cover this surgical specialty, should be adopted. (The name was changed to American Society of Oral Surgeons and Exodontists.) Several changes in the constitution and by-laws should also be made to facilitate management of the business affairs of the Society. Your president also believes that this Society should adopt standard terms to apply to our work, and endeavor

to encourage the use of these more dignified terms at all times. It should be the ambition of each member to concur in this matter.

Now that this organization has nearly reached the numerical limit of membership, great care should be exercised in the admission of new members. The committee in charge of this matter should take due care in their invitations to membership. It should be the future policy to admit only those who are willing to give to "the good of the cause," rather than those who may wish to have the honor of being members of this organization, simply to receive from it, and to give nothing in return.

This organization has now reached a place where it should be a power for good to mankind and one of the spokes in the wheel of science; but, in order to attain this great purpose, we must have before us at all times the aim to work for "the good of the cause," when we are called upon to "do our share." We should not be like the sponge, ever ready to absorb; but like the mountain spring, always ready to give freely. Harmony must prevail, if we are to succeed. Should any enter our ranks who may not be in sympathy with plans for the upbuilding and advancement of this organization, let us get rid of them, and, in their places, elect the right sort, who will put their shoulders to the wheel and help us all to pull together.

This Society should adopt a slogan to express our animating purpose. As a suggestion, I quote the following, from an article in the June issue of *Mentor*, by Edward W. Bok, editor and author (1921). "Ask a hundred men to choose the greatest word in the English language, and it is hardly likely that any two will agree. And yet, if we scan the present horizon of political, economic, social, and moral problems, domestic or foreign; if we approach the language with the idea of selecting the greatest word in it, as adapted to present needs, this one word naturally rises from all the other thousands and stands out supreme. It is the one word which, functioning in its fullest sense, would set at rest the world's problems; and it is because of its tremendously vital import to the needs of the present that it becomes, to my way of thinking, the greatest word in the English language. The word is *service*. Not the service that serves only self, but the service that labors for the interests of others and thus becomes the finest and best service to ourselves. Scale the word as you like, let it run the gamut of life in all its phases, and it holds its high place in the lives of men."

PROPOSED STANDARDIZATION OF SOME DENTAL TERMS AND PHRASES

REPORT OF THE COMMITTEE ON NOMENCLATURE, AMERICAN DENTAL ASSOCIATION[1]

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Your Committee begs to report as follows:

The purpose of the nomenclature of dentistry as of any profession is to provide the means for the intelligible interchange of ideas, to the end that its development and growth may progress and keep pace with that of the other professions.

Through its literature each profession becomes acquainted with the state of development of its sister professions and thereby is judged as to its intellectual status and the verity of its accomplishments. The scientific status, the exactness of knowledge, the cultural developments and the mental habits of a profession are distinctly reflected in its literature, and the retarding influence of insufficient and defective vehicles of expression must be removed if it is to keep pace with the other learned professions.

We are all conscious of the fact that the development of dentistry for the past two decades has been moving forward with such rapid strides, that our present terminology no longer meets the demands of the science and imposes a serious handicap upon our progress that we can ill afford longer to ignore.

The expansion of the field of dental activities resulting from the general recognition of the interrelationships of oral infections and bodily disease has necessitated an equivalent increase in our descriptive nomenclature. In response to this need for a larger terminology we have unfortunately been flooded with a group of terms that are manifestly amateurish in conception and defective in their etymology, hence they fail correctly to function as descriptive designations.

[1] Adopted by the House of Delegates of the American Dental Association, during the annual meeting of the Association in Los Angeles, California, July 17–21, 1922. Released, for publication in the September issues of dental journals, with the approval of the Secretary of the American Dental Association.—(W. J. G.)

It is quite apparent that the busy practitioner is indifferent to this important phase of our literature, seemingly being content with and almost demanding that the subject be dealt with by those intimately concerned with the historical record of dentistry in the literature, namely, teachers, writers, editors, etc., they being in a better position to undertake the task involved in the harmonizing of our present terminology and enlarging it to meet our requirements.

Since the notable efforts of Black, Guilford, Molyneaux, Wilson and others at the time of the World's Columbian Dental Congress, and later those of the American Institute of Dental Teachers, little has been done to increase and enlarge our nomenclature, with the exception of some individual efforts. Individual efforts, while they may be praiseworthy and often productive of much good, inevitably lead to confusion in the use of several words to mean the same thing, and mainly serve to impress more forcibly the necessity for coördination of efforts to the desired end.

Any effort, however, to standardize our nomenclature should be made with a definite purpose of conforming it as closely as possible to the general laws of nomenclature as already accepted by the biological sciences. The desirability of this course needs only to be mentioned here; so also is it only necessary to suggest the resulting enormous saving of duplication of work in the elemental phase of the undertaking that would accrue from this course.

There is a twofold responsibility involved in the adoption of a scientific dental terminology. Terms must not only express their meaning with precision, but, as in medicine, many terms are used to express a relation to a pathological or other biological phenomenon. These terms must not only be correct in an etymological sense; they must be so coined as to have a correct scientific meaning, and those who originate them must not only possess the cultural fundamentals necessary to constructive work in the science of nomenclature, but must also have a broad scientific vision, as well as an intimate knowledge of the subject in all its aspects.

Realizing the desirability of coördinating the various efforts being made to bring about uniformity of dental terms, and conscious of the necessity for a distinct forward step in the field of dental nomenclature, the Dental Editors' Club, an organization composed of the editors of dental magazines of the United States and Canada, at its meeting in Milwaukee passed the following resolution:

WHEREAS, The Dental Editors' Club of North America at its meeting in Milwaukee, August 17, 1921, realizes the pressing need for standardization of dental terms, be it

Resolved That the Dental Editors' Club of North America petition the House of Delegates of the National Dental Association to appoint a standing committee on nomenclature, to whom matters relating thereto emanating from various committees on nomenclature of other organizations be referred for consideration, to the end that the standardization and harmonizing of our technical dental terms may be under the direction and control of our National organization.

Pursuant to the intents and purposes of the above resolution, the National Dental Association appointed the following Committee on Dental Nomenclature: Drs. C. N. Johnson, Otto U. King, H. E. Friesell, H. L. Wheeler, and L. Pierce Anthony, as Chairman.

Soon after the adjournment of the Milwaukee meeting, the Chairman of the Committee took steps to get in close touch with those who have shown interest in this phase of dentistry, with the result that many suggestions from individual members of the profession were offered as to the adoption of new words and many criticisms made upon words now in use. We also have had the coöperation of several subordinate organizations of the National Dental Association who have suggested words relating particularly to the specialties with which they are concerned.

The Committee, while feeling the necessity for prompt action in regard to some of the words and suggestions offered, does not feel that it can present a final and definite report on all the terms that have been considered at this time.

It is not an easy task to decide upon the adoption of a certain class of words in dental nomenclature, as the conditions are continually changing; and it is practically impossible in some instances to foresee all the difficulties that may subsequently arise in the use of a word.

As an example one word will suffice. All are familiar with the long drawnout discussion of the use of the words "model" and "cast." After so many years the profession has about accepted the word "cast" as preferable to "model," as the word "model" is incorrect in the sense in which it has been so long used in dental literature. Now that "cast" has been accepted, the development of the method of inlay casting has much confused the use of the word "cast," so that now it is quite difficult in some instances to apply the word generally.

There is also much unforeseen difficulty in other phases of dental literature, namely, conforming our nomenclature to that of the other biologic sciences. We cannot afford to disregard the nomenclature of other sciences in forming our own, and while there are instances in which we have by determined effort succeeded in establishing the use of some words which have conflicted

with their use in other sciences, we do not feel that it is worth the effort and the resulting confusion consequent thereto. As examples of the latter, we might cite such words as "articulate," "cuspid," "bicuspid," "mandible," etc. In the case of the last-mentioned word it has caused much confusion in anatomical nomenclature for the reason that the nomenclature of the teeth, jaws and surrounding parts has been built around the word "maxillary" applied to both jaws, and the adaptation of mandible has been difficult and thus far not accomplished completely in relation to the anatomical terms given to the parts contiguous to the mandible.

All of this may, however, be avoided if we keep to the suggestion in the forepart of this report, namely, that our nomenclature be designed to conform to that of the other biologic sciences.

The objection is often raised to so-called hybrid words, *i.e.*, words which have both Greek and Latin derivative root words. This occurs to the Committee as being more or less pedantic. The vast majority of the words of our language are of Greek and Latin origin, and such being the case there is no valid reason apparent to the Committee why, if the two languages are chosen and preferred as derivative languages, we should not avail ourselves of the advantages to be gained by a combination of the two in forming our words.

Generalizations with regard to nomenclature, its purposes and the best methods of deciding upon terms are all well and good as suggesting principles upon which to work, but we realize that what is most desired particularly is some concrete result reached by the Committee. We have therefore and with careful deliberation prepared a list of words which we recommend to the Association for adoption and use in the sense in which we suggest they should be used.

We also present some words suggested which do not seem to conform to the intents and purposes of dental nomenclature, and which we recommend be abandoned as promptly as possible.

LIST OF WORDS RECOMMENDED BY THE COMMITTEE

alveolectomy (*L. alveolus* + *Gr. ektome*, excision). Excision of a portion of the alveolar process.

alveolotomy (*L. alveolus* + *Gr. tome, temnein*, to cut). Incision into the alveolus of a tooth, as for locating the end of a root of a tooth.

anesthesia. Preferable to *anaesthesia.*

apicoectomy [*L. apex*, gen. *apicis*, the end (of a tooth root) + Gr. *ektome*, excision]. The operation of excising the end of the root of a tooth. To be used in preference to *apectomy; apiectomy; apicectomy.*

artificial denture. Preferable to *plate.*

cuspid. To be used in preference to *canine.*

cementum. To be used in preference to *cement.*

conduction. To be used in preference to *conductive*, as in conduction anesthesia.

deciduous (adj.). To be used as designating the teeth of the first dentition, in preference to the terms, *temporary, milk* or *baby.*

dentural (adj.). (*L. dens, dentis*, tooth.) Relating to the denture.

first molar. To be used in preference to *six-year molar*, or *sixth-year molar.*

mandible (*L. mandibula* from *mandere*, to chew). The lower jaw.

maxilla, pl. *maxillæ* (*L. maxilla, jaw*). The upper jaw.

morsal and **occlusal** (adj.). To be used synonymously as relating to the masticating surfaces of the bicuspid and molar teeth.

centric occlusion. To be used to express the relation of the inclined planes of the teeth when the jaws are closed in the position of rest.

eccentric occlusion. To be used to express the relation of the inclined planes of the teeth in the excursive movements of the mandible.

mesial and **distal.** These terms as used today have been objected to as not being in conformity with anatomical nomenclature, where they are used to indicate relation to the median line of the body. They have, however, become so fixed in dental nomenclature that we do not suggest any change.

pathodontia (Gr. *pathos*, disease, + *odous*, tooth). That branch of dentistry which has for its purpose the study and treatment of diseases of the teeth.

pathology (Gr. *pathos*, disease, + *logos*, treatise). That branch of medical science which treats of morbid conditions, their causes, symptoms, etc. This term is being used to indicate a disease or pathologic condition, which is confusing, unnecessary, and undesirable.

pediadontia (Gr. *pais, paidos,* child, + *odous,* tooth). That branch of dentistry which has for its purpose the study and treatment of children's teeth and mouth conditions.

periodontia (Gr. *peri,* around, + *odous,* tooth). That branch of dentistry which has for its purpose the study and treatment of diseases occurring around the teeth and their roots.

periodontal (Gr. *peri,* around, + *odous,* tooth). Relating to the alveolodental ligament. To be used in preference to *peridental.*

periodontoclasia (Gr. *peri,* around, + *odous,* tooth, + *klassis,* a breaking down). The destructive degeneration of the tissues about the root of a tooth. Substituted for *pyorrhea alveolaris; Riggs' disease; interstitial gingivitis.*

periclasia (Gr. *peri, around,* + *klassis,* breaking down). Used as a shortening for convenience of periodontoclasia. Should be used with a qualifying word, as in itself it does not mean anything in particular.

pontic (*L. pons, pontis,* a bridge). (Adj. and noun.) A substitute for a natural tooth. Used in preference to *dummy.*

bicuspid. To be used in preference to premolar.

prosthesis (*n.*) (Gr. *pros,* to, + *tithenai,* to place). Preferable to *prothesis* (because of the more definite application of the Greek preposition *pros,* as compared to *pro,* in this form).

prosthetic (n.). Preferable to *prothetics.* (For the same reason as in prosthesis.)

pulpless tooth. To be used in preference to "*dead tooth,*" "*devital tooth,*" "*devitalized tooth.*" In cases where there is a "vital" pulp in a tooth or a "non-vital" pulp, it should be so designated; *e.g.,* a tooth with a vital pulp, or a tooth with a non-vital pulp.

radiology (n.) (*L. radius,* ray, + Gr. *logos,* treatise). The science of radiant energy. To be used as the generic term to indicate radiant energy from whatever source.

radiogram (n.) (*L. radius,* ray, + Gr. *gramma,* a writing). The product or tangible result, as the film or the print thereof, of the radiographic process, actuated by radiant energy of whatever source.

radiograph (v.) (*L. radius,* ray, + Gr. *graphein,* to write). The act or process of making a radiogram.

radiography. The art of making radiograms.

radiopaque (*L. radius*, ray, + *opacus*, shady). Term applied to a substance that is impermeable to radiant energy.

radiolucent (*L. radius*, ray, + *lucere*, to shine). Term applied to substances that allow the passage of radiant energy light, but offer some resistance.

radioparent (*L. radius*, ray, + *parere*, to appear). Term applied to substances that freely transmit the light of radiant energy.

Roentgen ray. To be used in preference to *x-ray*, and only where the specific ray is indicated.

roentgenology. The study and use of the Roentgen ray in its application to medicine and dentistry.

roentgenography. The art of making roentgenograms.

roentgenogram. The shadow picture produced by the Roentgen ray on a sensitized film, or the print from the film.

roentgenograph (v.). The act of making a roentgenogram.

second molar. To be used in preference to *twelve-year molar*, or *twelfth-year molar*.

third molar. To be used in preference to *wisdom tooth*.

Vincent's infection. To be used to express the ulcero-membranous stomatitis caused by Vincent's spirillum and fusiform bacillus in preference to *Vincent's angina;* the latter being more applicable to the throat infection.

x-ray (n.). This word is used indiscriminately as noun and verb. It should not be used as a verb. The word Roentgen ray is preferable. It should also be used with the small x rather than with the capital letter X, if used at all.

odontalysis; penetology. These two words have been suggested, the first to mean the examination of the teeth, and the latter, the science of radiant energy. We see no justification for these two words either etymologically or otherwise.

The Committee is pleased to state that in the near future there will be available places of accessible record of the activities in the field of dental nomenclature that have not been open to the profession since the passing of Harris' Dental Dictionary. If the present plans mature as proposed

there will soon be issued no less than three dictionaries devoted to dentistry, namely, one compiled by Dr. W. B. Dunning, under the auspices of the American Institute of Dental Teachers; one compiled under the direction of Dr. Louis Ottofy of Chicago; and a third compiled by the Chairman of this Committee. The Committee and the profession can thus feel assured of a permanent continuing record of their activities along this line in the future.

In concluding the report your Committee earnestly solicits the coöperation of Committees on Nomenclature and of individuals who are actively interested in this subject, to the end that our nomenclature may be as expeditiously as possible enlarged to meet the needs of the profession.